A CROSS SECTIONAL STUDY OF METABOLIC SYNDROME IN 100 STROKE PATIENTS

Dissertation Submitted For
M.D. Degree in General Medicine
Branch-I

Dr. M.G.R Medical University
Chennai
March 2008

DEPARTMENT OF GENERAL MEDICINE
COIMBATORE MEDICAL COLLEGE & HOSPITAL
COIMBATORE

CERTIFICATE

This is to certify that the Dissertation entitled ***" A CROSS SECTIONAL STUDY OF METABOLIC SYNDROME IN 100 STROKE PATIENTS*** *herewith submitted by* ***Dr.M.RANJANEE,*** *Post Graduate in General Medicine, Coimbatore Medical College to the Tamilnadu Dr. M.G.R. Medical University, is a record of a bonafide research work carried out by her under my guidance and supervision from August 2006 to August 2007.*

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DECLARATION

*I solemnly declare that the Dissertation titled "**A CROSS SECTIONAL STUDY OF METABOLIC SYNDROME IN 100 STROKE PATIENTS",** was done by me at Coimbatore Medical College & Hospital during the period from August 2006 to August 2007 under the guidance and supervision of Prof. Dr.K.Umakanthan,M.D.*

This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of M.D. Degree (Branch I) in General Medicine.

Place: Coimbatore ***Dr. M.RANJANEE***
Date: 20.11.07

ACKNOWLEDGEMENT

I sincerely thank ***Dr.HEMALATHA GANAPATHY, M.D.,*** *Dean C.M.C Hospital, and Coimbatore for having permitted me to undertake the study in this prestigious institution.*

It is a great pleasure to express my sincere thanks to ***Prof.Dr.UMAKANTHAN, M.D.,*** *my Unit Chief & Head of the Department of Medicine, C.M.C Hospital, Coimbatore for his able stewardship in the preparation of this work.*

I gratefully acknowledge my indebtedness to ***Prof.*** *Capt.* ***K.GOVINDRAJAN, M.D., D.M.,*** *Professor& HOD of Neurology, Coimbatore for his valuable guidance in the preparation of this dissertation work.*

I gratefully acknowledge the cooperation, goodwill and assistance rendered to me by the departments of Biochemistry, Microbiology and Radiology during the course of my study.

I whole heartedly thank my unit Assistant Professors viz. ***Dr.G.SELVARAJ, M.D. Dr.R.MANOHARI, M.D., Dr. S. AVUDAIAPPAN, M.D. & Dr.T.GEETHA, M.D.*** *for their professional assistance in shaping out this dissertation work.*

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INTRODUCTION

Stroke is a leading cause of death and disability in developing countries, afflicting individuals at all ages. Diabetes, being a major risk factor for stroke and with its increasing prevalence in India, has contributed alarmingly to the increase in the prevalence of stroke rate. Metabolic syndrome, a clustering of disturbed glucose and insulin metabolism, obesity and abdominal fat distribution, dyslipidemia, and hypertension is gaining recognition as an important independent risk factor for vascular disease and stroke. Because of its strong association with diabetes, it is often considered as a prediabetic condition.

Foreign studies have shown that men with the condition have a 78 percent greater stroke risk and similarly women with the condition have more than double the stroke risk of women who do not have the syndrome. Also it is shown that treating the risk-factor components of metabolic syndrome might reduce stroke risk before the onset of Type 2 diabetes. However, the contribution of its established components to ischemic stroke in adults has not been evaluated systematically in Indians. So it was proposed to examine the relationship of metabolic syndrome, as defined by modified National Cholesterol Education Program (NCEP) criteria with the risk for stroke.

DEFINITION OF METABOLIC SYNDROME ACCORDING TO THE WHO, NCEP ATP III, AND IDF

Factor	WHO (main criterion+ 2 factors)*	NCEP ATP III (any combination of 3 factors)	IDF (main criterion + 2 factors)
BMI (kg/m^2)	>30	—	—
Abdominal obesity (men/women)	Waist-to-hip ratio >0.9/0.85	Waist >102/88 cm	**Waist >94/80 cm**†
Triglycerides (mg/dl)	≥150	≥150	≥150
HDL cholesterol (mg/dl) (men/women)	<35/39	<40/50	<40/50
Blood pressure (mmHg)	≥140/≥90	≥130/≥85	≥140/>90 or >130/>85
HOMA‡	**>4.3**	—	—
Type 2 diabetes	**Present**	—	Present
Fasting glucose (mg/dl)	**≥110**	≥110	≥100
Fasting insulin	—	—	—
Urinary albumin excretion	≥20 µg/min or ≥30 mg/g albumin creatinine ratio	—	—

WHO- World Health Organization,

NCEP ATPIII-The National Cholesterol Education Program Adult Treatment Panel III (2001)

IDF- International Diabetes Federation

HOMA- Homeostasis model assessment, as a simple method for determining insulin sensitivity in humans; calculated as Fasting insulin ×fasting glucose (mmol/L) / 22.5

BMI- Body mass index

- Sines qua nonfactors are in bold.
- * According to the WHO, either BMI or abdominal obesity represents one criterion.
- †Ethnic group waist circumference (as measure of central obesity): Europid men ≥94 and women ≥80 cm; South Asian men ≥90 and women ≥80 cm; Chinese men ≥90 and women ≥80 cm; and Japanese men ≥85 and women ≥90 cm. Ethnic South and Central American populations use South Asian recommendations until more specific data are available. Sub-Saharan African populations use European data until more specific data are available. Eastern Mediterranean and Middle East (Arab) populations use European data until more specific data are available.
- ‡According to the WHO, HOMA, type 2 diabetes, and fasting glucose are alternatives, fulfilling one criterion.

Aim of the Study

AIM OF THE STUDY

1) To study the prevalence of metabolic syndrome in 100 stroke patients.

2) To study the role of various components of metabolic syndrome (modified ATP III criteria) in causation of stroke.

3) To assess the association between the metabolic syndrome and cerebrovascular accidents.

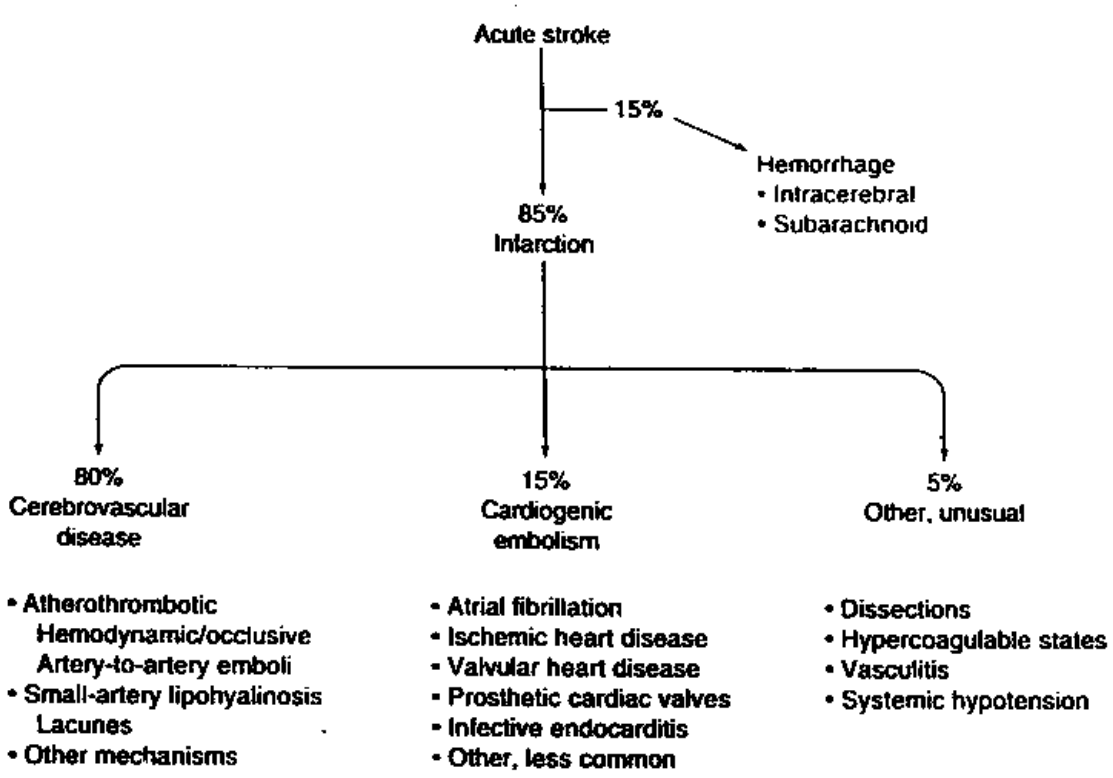
Review of Literature

REVIEW OF LITERATURE

STROKE

World Health Organization has defined stroke[1] as "neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours".

CLASSIFICATION OF CEREBROVASCULAR DISEASE[3]



Types of stroke[2]

i) Ischemic - can be due to thrombosis, embolism, or systemic hypoperfusion (Watershed or Border Zone stroke), or venous

thrombosis. Cocaine abuse doubles the risk of ischemic strokes[4]. 80% of strokes are due to ischemia.

ii) Hemorrhage - can be due to intracerebral hemorrhage, subarachnoid hemorrhage, subdural hemorrhage, or epidural hemorrhage. Amphetamine abuse quintuples and cocaine abuse doubles the risk of hemorrhagic strokes[5].

Thrombotic stroke

In thrombotic stroke, a thrombus-forming process develops around atherosclerotic plaques in the affected artery which gradually narrows the lumen of the artery and impedes blood flow to distal tissue. Since blockage of the artery is gradual, onset of symptomatic thrombotic strokes is slower. A thrombus itself (even if non-occluding) can lead to an embolic stroke if the thrombus breaks off-at which point it is then called an "embolus." Thrombotic stroke can be divided into two types depending on the type of involved vessel:

a) **Large vessel disease -** involves the common and internal carotids, vertebral, and the Circle of Willis. Causes include:

- Atherosclerosis
- Dissection
- Takayasu & Giant cell arteritis
- Moyamoya syndrome &Fibromuscular dysplasia

b) **Small vessel disease** - involves the intracerebral arteries, branches of the Circle of Willis, middle cerebral artery, stem, and arteries arising from the distal vertebral and basilar artery.

- Lipohyalinosis (lipid hyaline build-up secondary to hypertension and aging) and fibrinoid degeneration (stroke involving these vessels are known as lacunar infarcts)
- Microatheromas from larger arteries that extend into the smaller arteries (atheromatous branch disease)

Embolic stroke[2]

It refers to the blockage of arterial access to a part of the brain by an embolus—a traveling particle or debris in the arterial bloodstream originating from elsewhere which could either be a blood clot, a ruptured plaque or fat (e.g., from bone marrow in a broken bone), air, infected particle and even cancerous cells.

Because the embolic blockage is sudden in onset, symptoms usually are maximal at start. Also, symptoms may be transient as the embolus lyses and moves to a different location or dissipates altogether. Common causes include[4]:

- Atrial fibrillation and Sustained atrial flutter
- Rheumatic mitral or aortic valve disease
- Bioprosthetic and mechanical heart valves

- Atrial or ventricular thrombus; Papillary fibroelastoma & Left atrial myxoma
- Sick sinus syndrome
- Recent myocardial infarction (within one month) &Coronary artery bypass graft (CABG) surgery
- Symptomatic congestive heart failure with ejection fraction <30 percent and Dilated cardiomyopathy
- Libman-Sacks, Infective & Marantic endocarditis
- Antiphospholipid syndrome
- Complex atheroma in the ascending aorta or proximal arch

Systemic hypoperfusion (Watershed stroke)

Systemic hypoperfusion is the reduction of blood flow to all parts of the body including brain especially "watershed" areas --- border zone regions supplied by the major cerebral arteries. It is most commonly due to

1) cardiac pump failure from cardiac arrest or arrhythmias, or from reduced cardiac output as a result of myocardial infarction, pulmonary embolism, pericardial effusion, or bleeding.
2) Hypoxemia.

Hemorrhagic stroke[2]

A hemorrhagic stroke, or cerebral hemorrhage occurs when a blood vessel in the brain ruptures or bleeds. This interrupts the brain's blood

supply and if the bleeding continues, it can cause increased intracranial pressure. In addition, blood irritates brain tissue, disrupting the delicate chemical balance. In this respect, hemorrhagic strokes are more dangerous than their more common counterpart, ischemic strokes.

ISCHEMIC/EMBOLIC STROKE

A. ANATOMY[3]

1. Carotid Artery distribution-carotid arteries perfuse the majority of the cerebrum. Common Carotid Artery on either side divides into the Internal Carotid Artery and the External Carotid Artery. Internal Carotid Artery then divides into Anterior Cerebral Artery (ACA) and the Middle Cerebral Artery (MCA).

a. ACA-supplies the medial surface of the frontal lobe, parietal lobe and occipital lobe

b. MCA-the largest branch of the internal carotid artery

2. Vertebrobasilar Artery distribution-perfuses the base of cerebrum and majority of cerebellum.

   Two Vertebral Arteries join to form the Basilar Artery. Branching from the Basilar Artery are the 2 Posterior Cerebral Arteries (PCA).

a. Basilar Artery and PCA supply the occipital lobe, brain stem and cerebellum.

Fig - 1 ANTERIOR CEREBRAL ARTERY

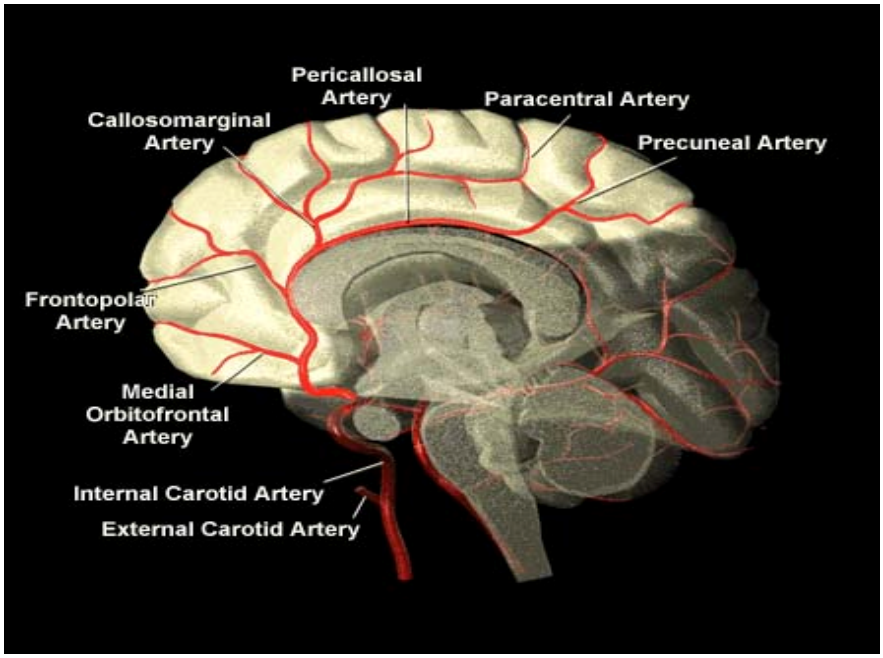


Fig - 2 MIDDLE CEREBRAL ARTERY

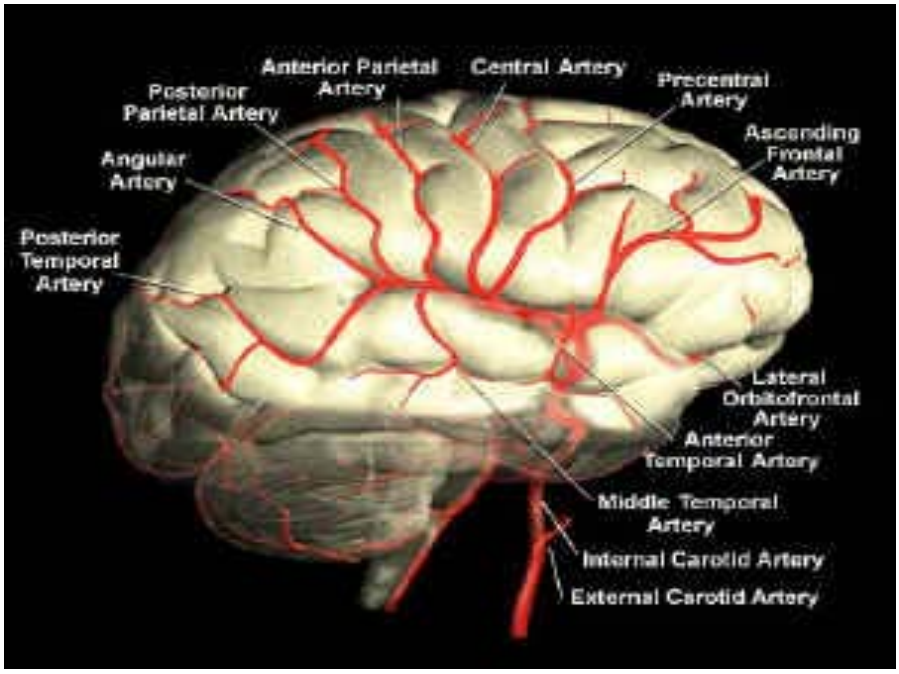


Fig - 3 POSTERIOR CEREBRAL ARTERY

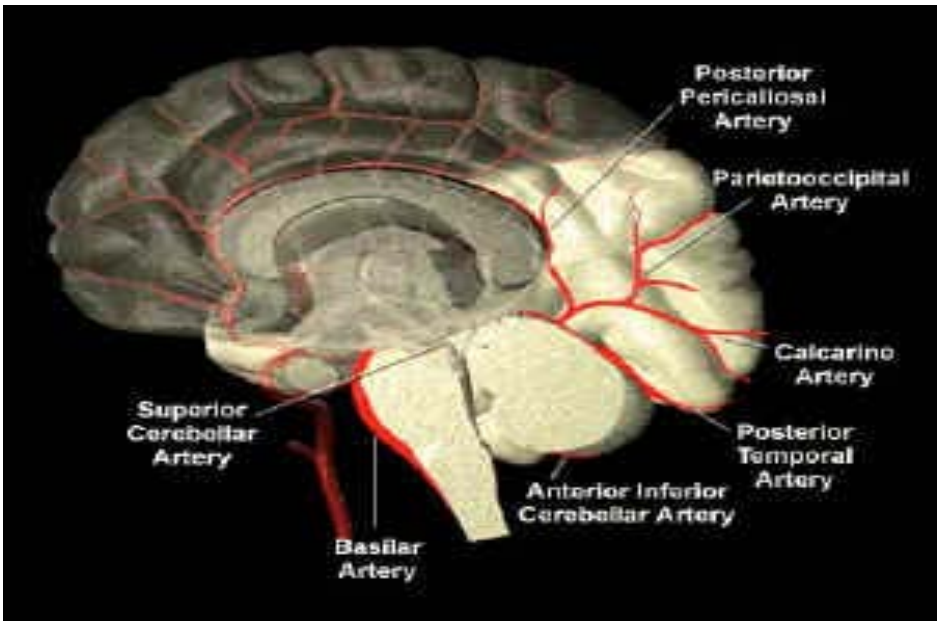


Fig - 4 ARTERIAL CIRCLE OF WILLIS

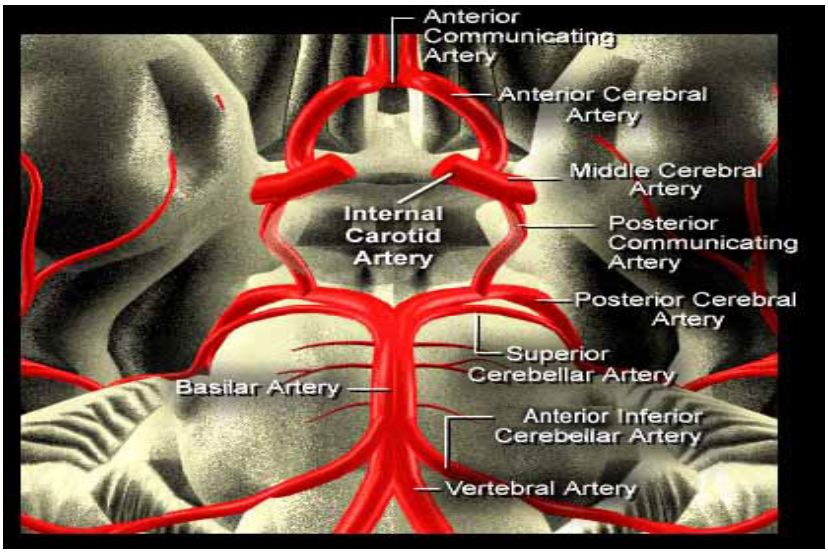
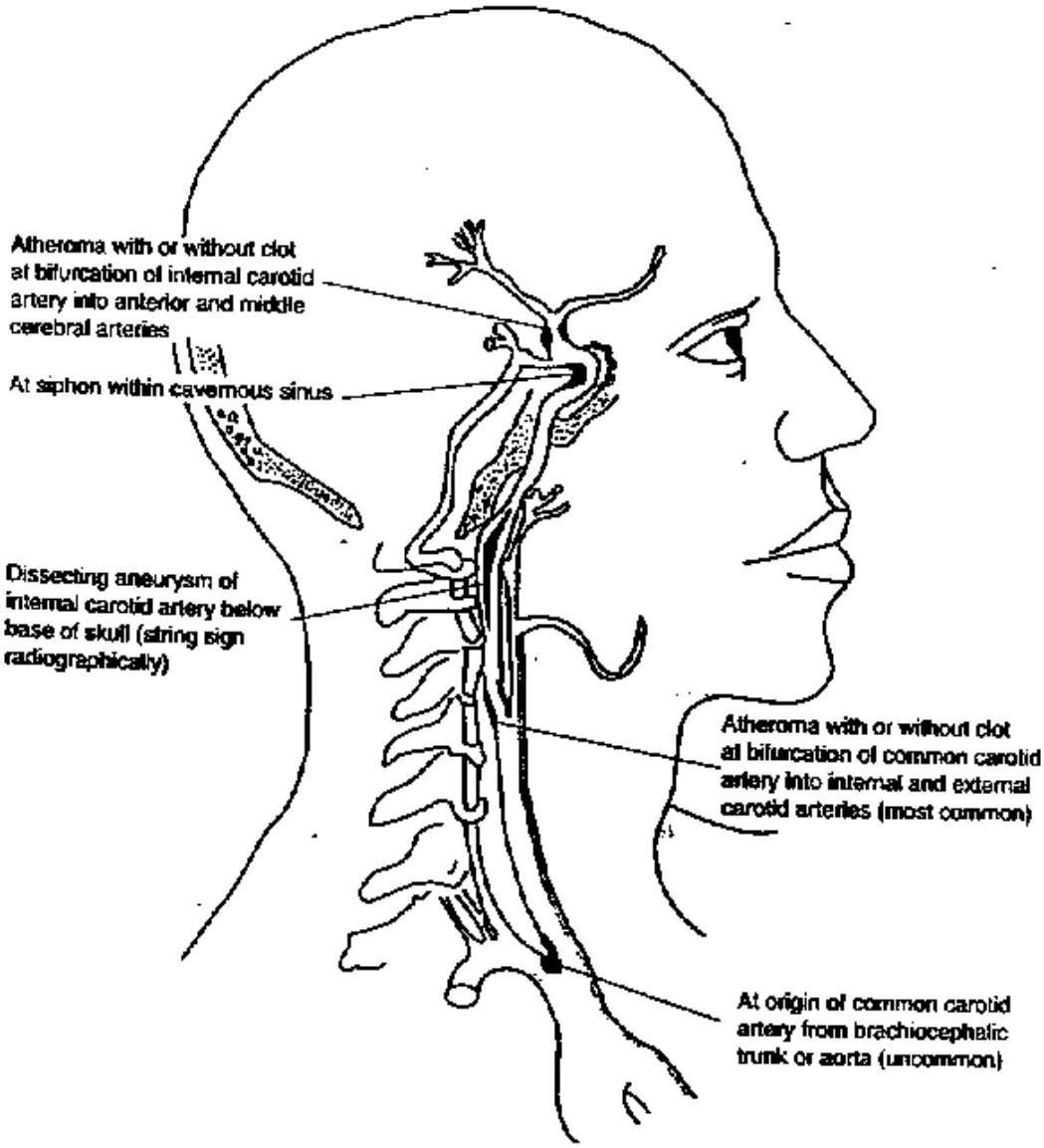


Fig.5 Various Sites of thrombo-embolic phenomena[3]



B. CLASSIFICATION OF ISCHEMIC EVENTS[3]

(These are based on the temporal course and eventual outcome).

1. Transient Ischemic Attacks (TIAs)

They are episodes of temporary reduction in perfusion to a focal region of the brain causing a short-lived disturbance of function. The

patient experiences a temporary focal neurological deficit such as slurred speech, aphasia, amaurosis fugax (monocular blindness), weakness or paralysis of a limb. The onset is rapid, usually less than 5 minutes and duration is usually 2-15 minutes but can last up to 24 hours. No neurological deficit remains after the attack & can vary from one episode in a lifetime to > 20 in one day. It may be the only warning of an impending stroke.

2. Reversible Ischemic Neurological Deficit (RIND)

Focal brain ischemia in which the deficit improves over a maximum of 72 hours and deficits may not completely resolve in all cases.

3. Cerebral Infarction

Permanent neurological disorder, the patient presents with fixed deficits; can present in 3 forms:

1. **Stable** - the neurological deficit is permanent and will not improve or deteriorate.
2. **Improving** - return of previously lost neurological function over several days to weeks.
3. **Progressing-** the neurological status continues to deteriorate following the initial onset of focal deficits, may see a stabilization period, followed by further progression.

C. PATHOPHYSIOLOGY

1. Atherosclerosis and subsequent plaque formation results in arterial narrowing or occlusion and predispose to thrombus formation.
2. Platelet Aggregation
   a. Vessel wall injury→ exposed sub endothelium→ triggers platelet activation
   b. Release of ADP from activated platelets causes platelet aggregation.
   c. Consolidation of platelet-plug by RBCs, coagulation factors, and formation of fibrin network.
   e. Thromboxane A2 (TX A2) produced by platelets and endothelium promote platelet aggregation and vasoconstriction.
3. Coagulation Cascade
   a. is a series of enzyme complexes located on the surface of platelets and endothelium which lead to thrombin production.
   b. Thrombin (IIa) then converts Fibrinogen to Fibrin.

Risk Factors for Stroke

- **Hypertension & Diabetes**
- Heart disease esp. atrial fibrillation
- Cigarette smoking
- Transient ischemic attacks

- **Increased serum cholesterol / lipids**
- Physical inactivity
- Obesity
- Excessive alcohol intake / drug abuse

METABOLIC SYNDROME

It is a prediabetic aggregation of symptoms and a more prevalent risk factor for stroke than is type 2 diabetes mellitus[6]. India is already the "Diabetes Capital" of the world and by the year 2020, is projected to have the highest number of individuals suffering from atherothrombotic cardiovascular disease[7].This concept was proposed to highlight the co-occurrence of risk factors for coronary heart disease (CHD) and type 2 diabetes. Subsequently, epidemiological studies have confirmed that this syndrome is common in a wide variety of ethnic groups including Caucasians, Afro-Americans, Mexican-Americans, Asian Indians, Chinese, Australian Aboriginals, Polynesians and Micronesians[8].

History

The term "metabolic syndrome" dates back to at least the late 1950s, but came into common usage in the late 1970s to describe various associations of risk factors with diabetes that had been noted as early as the 1920s[9, 10].

In 1977, Haller used the term "metabolic syndrome" for associations of obesity, diabetes mellitus, hyperlipoproteinemia, and hyperuricemia and steatosis hepatis when describing the additive effects of risk factors on atherosclerosis[11].

In 1988, in his Banting lecture, Gerald M. Reaven proposed insulin resistance as the underlying factor and named the constellation of abnormalities Syndrome X. Reaven did not include abdominal obesity, which has also been hypothesized as the underlying factor, as part of the condition.[12]

Various synonyms used are **Insulin resistance syndrome**, **Syndrome X, Reaven's syndrome** or **CHAOS** (Australia).Deadly quartet of central obesity, hypertension, dyslipidemia (high triglyceride and low HDL), and glucose intolerance is now a well-recognised entity.

The clinical manifestations of metabolic syndrome vary in different populations. Caucasians mainly show dyslipidemia, African populations show hypertension, Native Americans show hyperglycemia and South Asians show both hyperglycemia and accelerated coronary artery disease.[13]

ATP III[14] identified 6 components of the metabolic syndrome that relate to CVD:

- Abdominal obesity
- Atherogenic dyslipidemia
- Raised blood pressure
- Insulin resistance ± glucose intolerance
- Proinflammatory state
- Prothrombotic state

Abdominal obesity - most strongly associated with the metabolic syndrome. It presents clinically as increased waist circumference. Leptin deficiency or resistance leads to tissue deposition of fat. This ectopic distribution of fat (triglycerides) particularly its visceral or central component causes insulin resistance. Genetic factors are responsible for 70% of the variation in intra-abdominal fat mass. The other important determinant is sex, with males being typified by central and females by peripheral fat distribution.

Various theories put forth were:

1) "Common soil" theory: it is possible that visceral adiposity and metabolic disorders are not causally linked, but the end-result of common genetic and environmental factors.

2) Visceral adipocytes are metabolically more active and excess adipose tissue lipolysis leads to increased plasma concentrations of free fatty acids (FFA).
3) Because of anatomical proximity to liver, metabolic products of visceral adipocytes (mainly FFA) drain via the portal venous system directly to the liver[13].

There is strong evidence to suggest that excess FFA is the cause of insulin resistance. There is intracellular accumulation of triglycerides and fatty acid metabolites (fatty acid CoA's, diacylglycerol, and ceramides) in the insulin responsive tissues, interfering with upstream insulin signaling events in skeletal muscle & liver. Thus, insulin resistance in metabolic syndrome is post-receptor in origin.

Atherogenic dyslipidemia **-** is characterized by

- Increased triglyceride
- Decreased HDL cholesterol concentrations
- Normal concentrations of LDL cholesterol, but preponderance of small, dense LDL particles.

Other lipoprotein abnormalities eg, increased remnant lipoproteins, elevated apolipoprotein B, small LDL particles and small HDL particles have also been implicated as being independently atherogenic.

Increased hepatic FFA delivery leads to increased triglyceride synthesis and subsequently increased hepatic VLDL apoB-100 secretion which in turn increases the activity of cholesterol ester transfer protein (CETP) causing increased transfer of triglyceride from VLDL to LDL and HDL in exchange for cholesterol. These triglyceride-enriched lipo-proteins are good substrate for the enzyme hepatic lipase, which by hydrolysing the triglyceride causes:

(i) increased clearance of HDL cholesterol from blood, and

(ii) formation of highly atherogenic, small, dense LDL particles [15].

South Asian populations show lower muscle mass and high body fat at non-obese body mass index with tendency to central deposition of fat since childhood[16].

Elevated blood pressure - strongly associates with obesity and commonly occurs in insulin-resistant persons. Hypertension thus commonly is listed among metabolic risk factors. Leptin may increase blood pressure by causing sympathetic activation. Renal sodium retention secondary to hyperinsulinemia also contributes to hypertension.

Endothelial dysfunction manifesting as blunting of the biologic effect of a potent endothelium-derived vasodilator, nitric oxide, and increased production of vasoconstrictors such as angiotensin II, endothelin-1, cyclooxygenase and lipooxygenase products of arachidonic acid metabolism, seems to be the most important mechanism.

Insulin resistance - It is a physiological change in insulin action manifesting as resistance to insulin-mediated glucose disposal. It is the fundamental defect linking individual components of metabolic syndrome, although the strength of association of insulin resistance to these components is variable in different populations and even within populations. Patients with longstanding insulin resistance frequently manifest *glucose intolerance*, which itself is an independent risk factor for CVD[17].

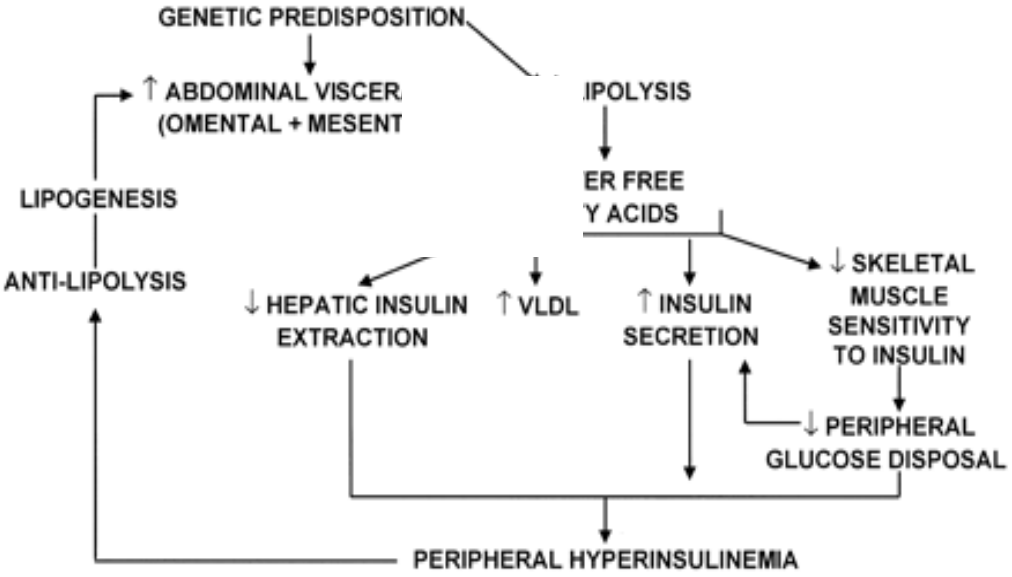
Measurement of insulin resistance[18, 19]

The euglycaemic-insulin clamp and the intravenous glucose tolerance test (IVGTT) are standard methods for the measurement of insulin resistance in research, but are impractical in clinical practice. Studies have shown fasting insulin of > 12.2 mU/L to be highly specific for insulin resistance. Also TGL /HDL-C concentration ratio is an equally powerful predictor of insulin resistance.

Abnormalities associated with insulin Resistance / hyperinsulinemia.

1. Glucose intolerance- Impaired fasting glucose &
   Impaired glucose tolerance
2. Abnormal uric acid metabolism- ↑Plasma uric acid concentration &
   ↓Renal uric acid clearance
3. Dyslipidemia - ↑ TGL &post prandial lipemia; ↓HDL-C, LDL

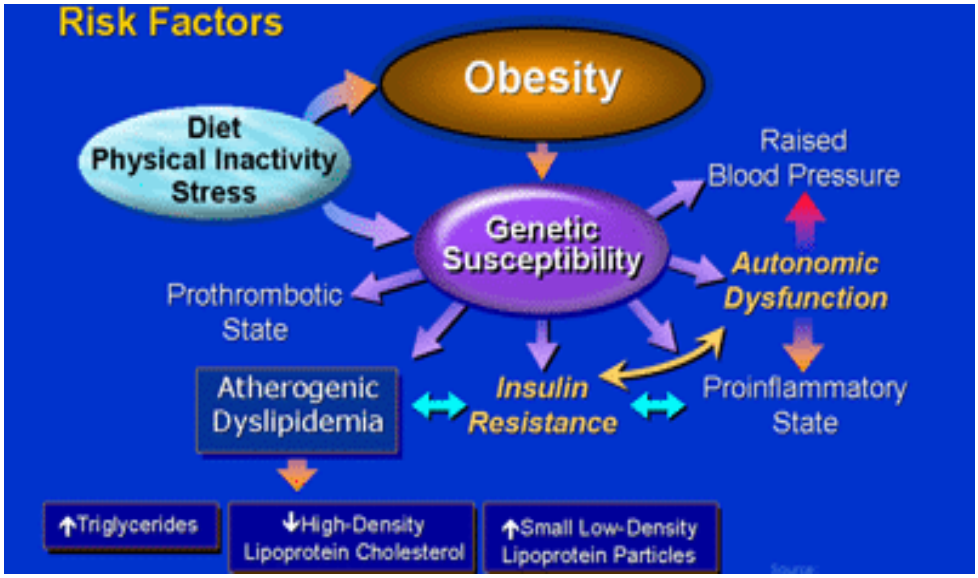
4. Hemodynamic - ↑ Sympathetic activity, Blood Pressure &
   Renal sodium retention
5. Haemostatic- ↑Plasma activator inhibitor-1 & Fibrinogen
6. Endothelial dysfunction - ↑Mononuclear cell adhesion
   ↑Plasma concentration of asymmetric
   dimethyl - arginine and
   ↓ Endothelial- dependent vasodilatation
7. Reproductive - Polycystic ovary syndrome.



A ***proinflammatory state*** **-** recognized clinically by elevated C-reactive protein (CRP), is commonly present in persons with metabolic syndrome. It is related to obesity, because excess adipose tissue releases inflammatory cytokines causing elevated CRP levels.

A ***prothrombotic state*** - characterized by increased plasma plasminogen activator inhibitor (PAI)-1 and fibrinogen, also associates with the metabolic syndrome. Thus, prothrombotic and proinflammatory states may be metabolically interconnected.

Fig 6 Clustering of various risk factors in metabolic syndrome



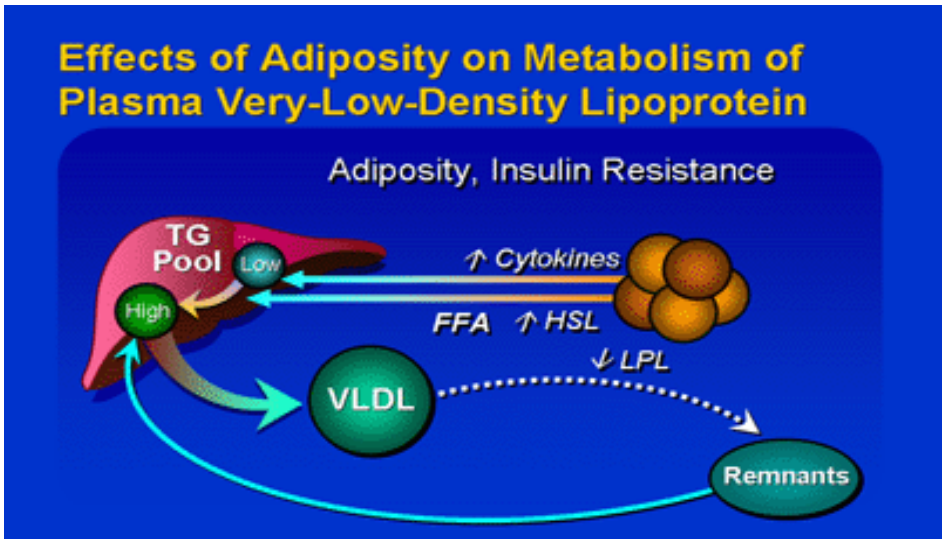
Pathogenesis of Metabolic Syndrome

a) Obesity and Abnormal Body Fat Distribution

Obesity contributes to hypertension, dyslipidemia and hyperglycemia. Abdominal obesity especially correlates with metabolic risk factors. Excess adipose tissue releases several products like nonesterified fatty acids (NEFA), cytokines, PAI-1 and adiponectin which overload muscle and liver with lipid, thereby enhancing insulin

resistance. High CRP levels and low adiponectin levels accompanying obesity may signify a proinflammatory state. An elevated PAI-1 contributes to a prothrombotic state.

Fig 7



HSL-hormone sensitive lipase; LPL- lipoprotein lipase, TG-triglyceride and VLDL-very low density lipoprotein.

b) **Insulin resistance**

c) **Other Contributing Factors –**

1) **Advancing age -** The prevalence of metabolic syndrome increases with age, affecting less than 10 percent of people in their 20s and 40 percent of people in their 60s[20].

2) **Race -** Hispanics and Asians seem to be at greater risk for metabolic syndrome than other races.

3) **Elevated C - Reactive protein**- Obesity and the metabolic syndrome are associated with elevated levels of CRP, suggesting a strong relationship between the metabolic derangement and inflammation. Studies have shown that elevated CRP increased progressively from 1.9 for those with one risk factor to 6.8 when all five of the ATP III criteria were present.

4) **Plasminogen activator inhibitor-1 (PAI-1)** is a protein released into the circulation from endothelial cells that inhibits tissue-type plasminogen activator (t-PA) which in turn leads to decreased plasmin (fibrinolysin) and ultimately, to increased levels of circulating fibrinogen. Both PAI-1 and fibrinogen correlate with levels of insulin and its precursors. The analysis of PAI-1 is not performed in most clinical laboratories[21].

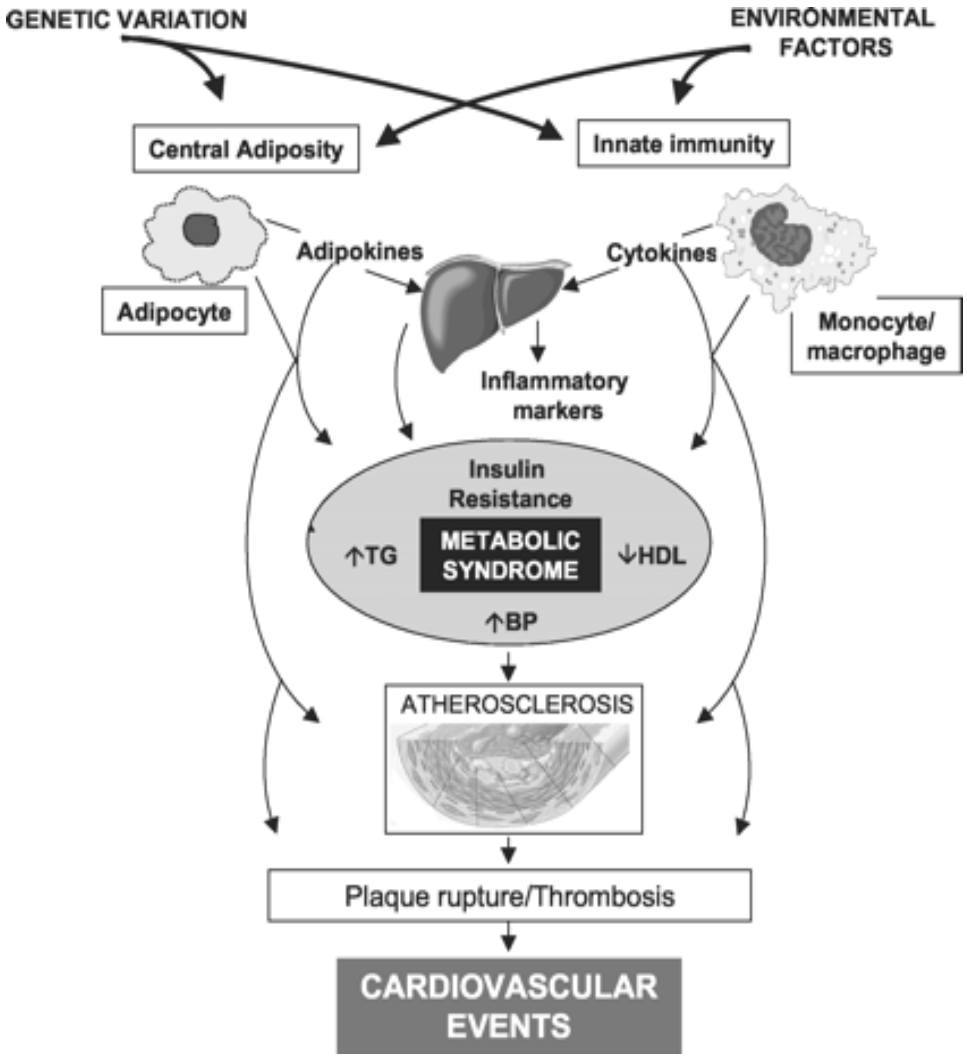
5) **Microalbuminuria**- an early indicator of nephropathy in both diabetic and hypertensive individuals and regarding metabolic syndrome, the WHO recommendation of $\geq$ 20µg/min for timed collections is in accordance with the definition of microalbuminuria[22].

6) **Uric acid** has been shown to be a powerful risk marker for CVD.

7) **Serum gamma - glutamyl transpeptidase, fibrinogen, factor - VIII: c, cytokines IL-6 & 10, TNF-α, adhesion molecules ICAM 1 & VCAM 1.**

8) **Fasting homocysteine levels.**

Fig 8 Pathogenesis of Cerebro- / Cardio vascular events in Metabolic Syndrome



&

CEREBROVASCULAR EVENTS

Criteria for Clinical Diagnosis of Metabolic Syndrome

a) EGIR

European Group for the Study of Insulin Resistance (EGIR) (1999) requires insulin resistance defined as the top 25% of the fasting insulin values among non-diabetic individuals AND two or more of the following:

- **central obesity**: waist circumference ≥ 94 cm (male),
  ≥ 80 cm (female)
- **dyslipidemia**: TG ≥ 2.0 mmol/L and/or
  HDL-C < 1.0 mg/dL or treated for dyslipidemia
- **hypertension**: blood pressure ≥ 140/90 mmHg or
  antihypertensive medication
- **fasting plasma glucose** ≥ 6.1 mmol/L

b) NCEP ATP III[14]

The National Cholesterol Education Program Adult Treatment Panel III (2001) requires at least three of the following:

Risk Factor	Defining Level
1. Abdominal obesity, given as waist circumference	
Men	>102 cm (>40 in)
Women	>88 cm (>35 in)
2. Triglycerides	≥150 mg/dL

3. HDL cholesterol

Men	<40 mg/dL
Women	<50 mg/dl

4. Blood pressure ≥130/≥85 mm Hg

5. Fasting glucose ≥110 mg/dL[‡]

 Waist circumference is measured at the midpoint between the lower border of the rib cage and the iliac crest. It is simpler to measure, culturally more acceptable to our population and is a better indicator of central obesity compared to WHR[23].

‡ The American Diabetes Association has recently established a cutoff point of ≥100 mg/dL, above which persons have either prediabetes (impaired fasting glucose) or diabetes. This new cutpoint should be applicable for identifying the lower boundary to define an elevated glucose as one criterion for the metabolic syndrome.

c) World Health Organization

In 1998, a World Health Organization (WHO) consultation group outlined a working definition of the metabolic syndrome[24]. A potential disadvantage is the need for special testing of glucose status beyond routine clinical assessment.

Advantages:

i) Each component of the syndrome conveys increased cardiovascular risk, but as a combination they become much more powerful.

ii) The features of metabolic syndrome may be present for up to 10 years before detection of glycaemic disorders.

Insulin resistance, identified by one of the following:

- Type 2 diabetes [25]
- Impaired fasting glucos
- Impaired glucose tolera

Plus any 2 of the following:

- Antihypertensive medication and/or high blood pressure (≥140 mm Hg systolic or ≥90 mm Hg diastolic)
- Plasma triglycerides ≥150 mg/dL (≥1.7 mmol/L)
- HDL cholesterol <35 mg/dL (<0.9 mmol/L) in men or <39 mg/dL (1.0 mmol/L) in women
- BMI >30 kg/m^2 & /or waist: hip ratio >0.9 in men, >0.85 in women
- Urinary albumin excretion rate ≥20 µg/min or albumin:creatinine ratio ≥30 mg/g.

D) American Association of Clinical Endocrinologists[25]

These criteria appear to be a hybrid of those of ATP III and WHO metabolic syndrome.

Risk Factor Components	**Cut points for Abnormality**
❖ Overweight/obesity	BMI ≥25 kg/m^2
❖ Elevated triglycerides	≥ 150 mg/dL (1.69 mmol/L)
❖ Low HDL cholesterol	
Men	<40 mg/dL (1.04 mmol/L)
Women	<50 mg/dL (1.29 mmol/L)
❖ Elevated blood pressure	≥ 130/85 mm Hg

❖ 2-Hour post glucose challenge	>140 mg/dL
❖ Fasting glucose	110 -126 mg/dL
❖ Other risk factors	Family history of type 2 diabetes, hypertension, or CVD /cystic ovary syndrome Sedentary lifestyle Advancing age)ups having high risk for type 2 diabetes or CVD

26

E) AHA/NHLBI[26]

The American Heart Association and the National Heart, Lung, and Blood Institute updated the NCEP ATP III definition & recommended that the metabolic syndrome be identified as the presence of three or more of these components:

- **Elevated waist circumference:**

  Men — equal to or greater than 40 inches (102 cm)

  Women — equal to or greater than 35 inches (88 cm)

- **Elevated triglycerides**: Equal to or greater than 150 mg/dL

- **Reduced HDL ("good") cholesterol**:

  Men — Less than 40 mg/dL; Women — Less than 50 mg/dL

- **Elevated blood pressure**: ≥ 130/85 mm Hg or use of medication

for hypertension.

- **Elevated fasting glucose**: ≥ 100 mg/dL (5.6 mmol/L) or use of medication for hyperglycemia.

F) International Diabetic Fe 27)F) [27]

It defines metabolic sy ıe presence of **central obesity** (Waist circumference > 94 cm maıes ;> 80cm females) + any 2 of the following 4 factors:

Raised triglyceride level (>150 mg/dl),

Reduced HDL cholesterol (< 40males; <50 females),

Raised blood pressure (>130/85 mmHg) and

Raised fasting plasma glucose (>100mg/dl).

Advantage:

This definition has proposed ethnicity-specific cutoff values for waist circumference, namely, 94 and 80 cm for European men and women, respectively, and 85 and 90 cm for Japanese men and women, respectively. In contrast, NCEP ATP III criteria, applied to an Asian population will underestimate the population at risk. With a lower waist circumference cutoff, the prevalence of the metabolic syndrome is comparable to that in Western populations[28].

Metabolic Syndrome Features, Measurement of These Features, and Features Included in the Current NCEP ATP III Guidelines

Metabolic Syndrome Feature	Clinical Measures	Research Measures	NCEP ATP III Criteria
Obesity	Waist Body mass index	Displacement techniques Bioelectrical impedance DEXA scanning CT,MRI	Waist
Insulin Resistance	Fasting plasma glucose	Plasma leptin, adiponectin, resistin Plasma insulin	Fasting plasma glucose

	Oral glucose tolerance testing	HOMA/QUICKI Intravenous glucose tolerance testing Hyperinsulinemic clamp	
Hypertension	Systolic and diastolic BP Ambulatory BP	Vascular compliance/stiffness Microalbuminuria Angiotensin II Endothelin	Systolic and diastolic BP
Lipoproteins	TG, VLDL HDL Small dense LDL	Transfer proteins and enzyme activity Cholesterol efflux assays ex vivo Postprandial lipoprotein responses Lipoprotein turnover studies	TG HDL
Inflammation	Wh c C-reactive protein	Serum amyloid A Fibrinogen, factor VIII Sialic acid Cytokines IL-6, TNF-α, IL-10 Soluble adhesion molecules: ICAM-1,	NA

	VCAM, E-selectin	
Prothrombotic, fibrinolytic	Evoked inflammatory responses Plasma PAI-1, D-dimer Plasma FPA, F1–2	NA
	Urinary 11-dehydroTXB	NA
Oxidant stress	Oxidized LDL Isoprostanes	
Genetics	DNA /protein adducts Candidate gene single nucleotide polymorphism MetSyn (SNPs) Genome-wide scan—linkage analysis	NA

BP-blood pressure, **DEXA-**dual-energy X-ray absorptiometry

HOMA-, Homeostasis Model Assessment (HOMA)

QUICKI- Quantitative Insulin Sensitivity Check Index;

IL- interleukin, **TNF-** tumor necrosis factor,

ICAM, intercellular adhesion molecule,

VCAM-vascular cellular adhesion molecule

PAI- plasminogen activator inhibitor and **NA-** not applicable.

Therapeutic Goals and lations for Clinical Management of Metabolic Syndrome

Therapeutic Target and Goals of Therapy	Therapeutic Recommendations
Lifestyle risk factors	Long-term prevention of CVD and prevention (or treatment) of type 2 diabetes mellitus
Abdominal obesity Weight reduction by 7% - 10% during year 1 of therapy and achieve desirable weight (BMI <25 kg/m^2)	Weight maintenance/reduction through appropriate balance of physical activity, caloric intake, and formal behavior-modification programs when indicated to maintain/achieve waist circumference of <40 inches in men and <35 inches in women.
Physical inactivity Regular physical activity; at least 30 min of moderate-intensity continuous or intermittent at least 5 d/wk	In patients with established CVD, encourage 30 to 60 min of moderate-intensity aerobic activity: brisk walking, preferably daily, supplemented by increase in daily lifestyle activities (eg, pedometer step tracking, walking breaks at work, gardening, housework). Encourage resistance training 2 d/wk. Advise medically supervised programs for high-risk patients (eg, recent acute coronary syndrome or revascularization, CHF).
Atherogenic diet Reduced intake of saturated fat, transfat, cholesterol	Recommendations: saturated fat <7% of total calories; reduce *trans* fat; dietary cholesterol <200 mg/dL; total fat 25% to 35% of total calories. Most dietary fat should be unsaturated; simple sugars should be limited.
Metabolic risk factors **1. Atherogenic dyslipidemia**	Shorter-term prevention of CVD or treatment of type 2 diabetes mellitus

Primary target: ↑ LDL - C Secondary target: ↑non-HDL-C **High-risk patients**[*]: <130 mg/dL (3.4 mmol/L) (optional: <100 mg/dL [2.6 mmol/L] for very high-risk patients[†]**)** **Moderately high-risk patients**[‡]: <160 mg/dL (4.1 mmol/L) Therapeutic option: <130 mg/dL (3.4 mmol/L**)** **Moderate-risk patients**[§]: <160 mg/dL (4.1 mmol/L) **Lower-risk patients**[‖]: <190 mg/dL (4.9 mmol/L)	Reduce LDL-C according to ATP III guidelines. 1) to achieve non-HDL-C goal: Intensify LDL-lowering therapy. 2) Add fibrate (preferably fenofibrate) or nicotinic acid if non-HDL-C remains relatively high after LDL-lowering drug therapy (add fibrate or nicotinic acid in high-risk patients) 3) All patients: If TG ≥500 mg/dL, initiate fibrate or nicotinic acid (before LDL-lowering therapy; treat non-HDL-C)
Tertiary target: ↓ HDL-C No specific goal: Raise HDL -C to extent possible with standard therapies	Maximize lifestyle therapies: Weight reduction and increased physical activity. Add fibrate or nicotinic acid after LDL-C-lowering drug therapy
2. Elevated BP Reduce BP to <140/90 mm Hg (or <130/80 mm Hg if diabetes present). Reduce BP further to extent possible through lifestyle changes.	For BP ≥120/80 mm Hg: Initiate or maintain lifestyle modification in all patients with metabolic syndrome: weight control, increased physical activity, alcohol moderation, sodium reduction, and emphasis on increased consumption of fresh fruits, vegetables, and low-fat dairy products (DASH diet). For BP ≥140/90 mm Hg (or ≥130/80 mm Hg for individuals with chronic kidney disease or diabetes): As tolerated, add BP medication (ACE inhibitors, ARBs.)
3. Elevated glucose	
For IFG, delay progression type 2 diabetes mellitus.	'G, encourage weight reduction 'eased physical activity.

For diabetes, HbA_{1C} <7.0%	- For type 2 diabetes mellitus, lifestyle therapy& pharmacotherapy to achieve HbA1C (<7%). Modify other risk factors and behaviors.
4. Prothrombotic state Reduce thrombotic and fibrinolytic risk factors	High-risk patients: Initiate and continue low-dose aspirin therapy; - In patients with ASCVD, consider clopidogrel if aspirin is contraindicated. - Moderately high-risk patients: Consider low-dose aspirin prophylaxis
5. Proinflammatory state	Recommendations: no specific therapies beyond lifestyle therapies

***High-risk patients** - established ASCVD, diabetes, or 10-year risk for coronary heart disease >20%. For cerebrovascular disease, high-risk condition includes TIA or stroke of carotid origin or >50% carotid stenosis.

†**Very high-risk patients** - likely to have major CVD events in next few years, and diagnosis depends on clinical assessment. Factors that may confer very high risk include recent acute coronary syndromes, and established coronary heart disease + any of following: multiple major risk factors (especially diabetes), severe and poorly controlled risk factors (especially continued cigarette smoking), and metabolic syndrome.

‡**Moderately high-risk patients** - 10-year risk for CVD10% to 20%. Factors that favor therapeutic option of non-HDL-C <100 mg/dL are those that can raise individuals to upper range of moderately high risk: multiple major risk factors, severe and poorly controlled risk factors (especially continued cigarette smoking), metabolic syndrome, and documented advanced subclinical atherosclerotic disease (eg, coronary calcium or carotid intimal-medial thickness >75th percentile for age and sex).

§**Moderate-risk patients** - with 2+ major risk factors and 10-year risk <10%.

||**Lower-risk patients** are those with 0 or 1 major risk factor and 10-year risk <10%.

Elevated LDL-C: Primary Target of Lipid-Lowering Therapy in People at Risk for ASCVD [14]

Goals of Therapy	Therapeutic Recommendations
High-risk patients: <100 mg/dL *Very high risk patients*:<70 mg/dl	High-risk patients: lifestyle therapies. Add LDL-C-lowering drug if baseline LDL-C ≥100 mg/dL
Moderately high-risk patients: <130 mg/dL *High risk patients:* optional goal is <100 mg/dL (2.6 mmol/L)	Moderately high-risk patients: lifestyle therapies. Add LDL-lowering drug when LDL-C ≥130 mg/dL (3.4 mmol/L). If baseline LDL-C 100- 129 mg/dL, LDL-lowering therapy introduced if patient's risk is assessed to be in upper ranges of this risk category.
Moderate-risk patients: <130 mg/dL	Moderate risk patients: lifestyle therapies. Add LDL-C lowering drug when LDL-C ≥160 mg/dL (4.1 mmol/L).
Lower-risk patients: <160 mg/dL	Lower-risk patients: lifestyle therapies. Add LDL-C lowering drug when LDL-C ≥190 mg/dL after lifestyle therapies. For LDL-C 160 to 189 mg/dL, LDL- lowering drug is optional).

Materials and Methods

MATERIALS AND METHODS

This study was a cross-sectional study, conducted in 100 patients consisting of **70** males and **30** females with Cerebrovascular disease (Stroke) admitted in General wards at Coimbatore Medical College Hospital during the period of one year from August 2006 to August 2007.

The Inclusion criteria were

1) Both male and female patients presenting with neuro-radiological features of stroke (clinically and CT proven, including major and minor stroke).

The criteria used in the clinical diagnosis of stroke were those setforth by the Adhoc Committee of National Institute of Neurological diseases and blindness.

The **clinical diagnosis** was made by

a) eliciting a **detailed history** from the patient or from their relatives, regarding the nature of illness with special emphasis on the following:

i) mode of onset – sudden or gradual

ii) time of onset – during early morning hours soon after getting up or during day time activities.

iii) associated symptoms – headache, vomiting, convulsion, past history of Transient Ischemic attack (TIA), Diabetes mellitus, Ischemic heart disease, personal habits like cigarette smoking and alcohol drinking in both sexes and intake of oral contraceptive pills in the past in females.

b) **Physical examination** which included

i) General examination with special regards to obesity, carotid and peripheral pulsation, blood pressure, polycythemia, dehydration as predisposing factors and acanthosis nigricans as a marker of hyper insulinemia.

ii) Central nervous system examination for evidence of unconsciousness, hemiplegia or paresis, cranial nerve dysfunction, speech disturbance, sensory deficits, meningeal and cerebellar involvement and optic fundus examination.

iii) Cardio vascular respiratory and abdominal examination.

In all these patients, clinical diagnosis was confirmed by **CT scan of the brain** for designation of stroke as

a) Ischemic - patients with cerebrovascular thrombosis

b) Hemorrhagic – patients with cerebral hemorrhage

c) Unclassified – patients who were CT wise normal but had clinical evidence of stroke i.e. patients with minor stroke (Transient Ischemic Attack (TIA), Reversible Ischemic Neurological Deficit & Brain Stem stroke).

When there was suspicion of tumor or other etiology, the diagnosis was confirmed by MRI scan, Carotid Doppler, four vessel angiogram to rule out embolism, serum VDRL for syphilis, blood examination for anemia, renal failure, polycythemia, coagulation profile to rule out bleeding/coagulation defects as cause of cerebral hemorrhage. ECG, X-ray Chest and ECHO were done in relevant cases to rule out cardiovascular etiology.

2) The patients with age ranging from 30 years to 90 years were divided into 3 groups:

   a) up to 40 years ($\leq$ 40 years)

   b) 41- 59 years

   c) $\geq$ 60 years.

3) Patients presenting at the time of data collection.

The exlusion criteria were

i) patients with age less than 30 years.

ii) patients with embolic stroke, as there are no significant changes in the lipid profile in them.

iii) neurological deficit not fitting into above clinical /radiological criteria for cerebrovascular disease.

They were then subjected to a battery of tests that define metabolic syndrome according to one proposed by American Heart Association and

the National Heart Lung and Blood Institute, a modification of NCEP ACP- III definition. This definition was chosen as

a) it is clinically applicable in Indian context

b) relatively easy to perform, cost effective &

c) it specifies lower cut off points for waist circumference in Asians {males ≥ 90 cm (35 inches), females ≥ 80 cm (31 inches) }as compared to ≥ 102 cm in males and ≥ 88 cm in females respectively taken in ATP III criteria for metabolic syndrome.

These tests were –

i) **Fasting Blood Glucose (FBG) ≥ 100 mg/dl** or drug treatment for elevated glucose. It was measured by oxidase - peroxidase method. This test is specific, reproducible, sensitive and rapid.

Principle:

D-glucose+H_2O+O_2 $\xrightarrow[\text{(from aspergillus niger)}]{\text{Glucose oxidase}}$ → D-gluconic acid +H_2O_2

H_2O_2 + reduced O-Dianisidine $\xrightarrow[\text{(from horse radish)}]{\text{peroxidase}}$ → oxidized

(colorless) O-Dianisidine (Brown)

O-Dianisidine (Brown) $\xrightarrow{H2SO4}$ → oxidized O-Dianisidine (Pink)

The intensity of pink color measured at 540 nm is proportional to the original glucose concentration.

ii) **Triglyceride: ≥ 150 mg/dl in both sexes** were taken as fulfilling criteria for metabolic syndrome. It was measured by enzymatic method (Foster and Dunn) Hantzsh reaction.

Principle:

In this reaction, triglycerides are extracted by Haptane and saponified by potassium hydroxide. The glycerol is oxidized to formaldehyde which combines with acelylacetone in the presence of ammonium ions to give dihydrobutidine derivative which is measured calorimetrically at 415 nm.

iii) **High density lipoprotein (HDL) cholesterol** (Lopes and Virella)

< 50 mg/dl in females and **< 40 mg /dl in males.**

In this procedure, VLDL, Chylomicrons and LDL were precipitated by phosphotungstate in the presence of magnesium ions and the HDL cholesterol was estimated from the supernatant. All measurements were taken using highly sophisticated modular system of Boheinger Knoll, West Germany.

For both these tests, blood was withdrawn in the morning before breakfast after a minimum of 7 hours of fasting and collected in tubes without anticoagulant. Though fasting is not required for estimation of HDL cholesterol, as levels drop only slightly after fatty meal, it is important for estimation of exogenous lipids like chylomicrons and triglycerides.

In patients with concomitant acute myocardial infarction, lipid profile was done after 3 months as until such time the cholesterol level

may be lower than usual. In the case of patients with cerebral thrombosis, blood samples were taken 7 days after development of the neurological deficit. For patients with cerebral hemorrhage, blood samples were taken on the day of admission due to bad prognosis associated with them.

iv) **Waist Circumference: males ≥ 90 cm (35 inches)**
females ≥ 80 cm (31 inches)

It was measured halfway between highest level of iliac crest and lower rib margin during minimal inspiration.

v) **Blood Pressure - Systolic ≥ 130 mmHg** and
Diastolic ≥ 85 mmHg or antihypertensive drug therapy in a patient with history of hypertension.

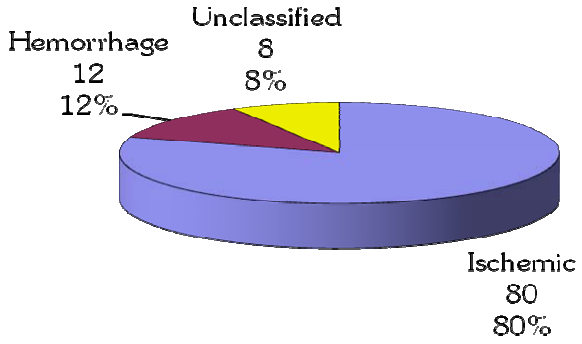
For this, 3 Blood Pressure readings were taken at 2 minute intervals in lying posture and highest value was taken into consideration, using a standard Sphygmomanometer.

Presences of any 3 of the above factors constitute the syndrome. Analysis was based on determining prevalence of metabolic syndrome in stroke patients and studying individual parameters in causation of stroke and assessing their percentage attributability to stroke.

Observation and Results

OBSERVATION AND RESULTS

Chart1: Stroke pattern in100 patients



Out of **100** Stroke patients studied, **80%** (80 patients) had Ischemic Stroke and **12%** (12 patients) had Hemorrhage stroke. Remaining **8%** (8 patients) were under Unclassified category i.e. clinically diagnosed as cerebrovascular disease but CT Brain reported normal. (Patients with Transient Ischemic Attack (TIA) /Reversible Ischemic Neurological Deficits (RIND) and cases of probable Brainstem stroke where CT Brain

Chart 2: Gender wise distribution of Stroke Pattern:

Out of **70** male stroke patients, **53** (76%) had Thrombotic stroke, **11** (16%) had cerebral hemorrhage and remaining **6** (8%) were under unclassified category.

Similarly in **30** female stroke patients, **27** (90%) had Thrombosis, **1** (3%) had hemorrhage and remaining **2** (7%) were under unclassified category.

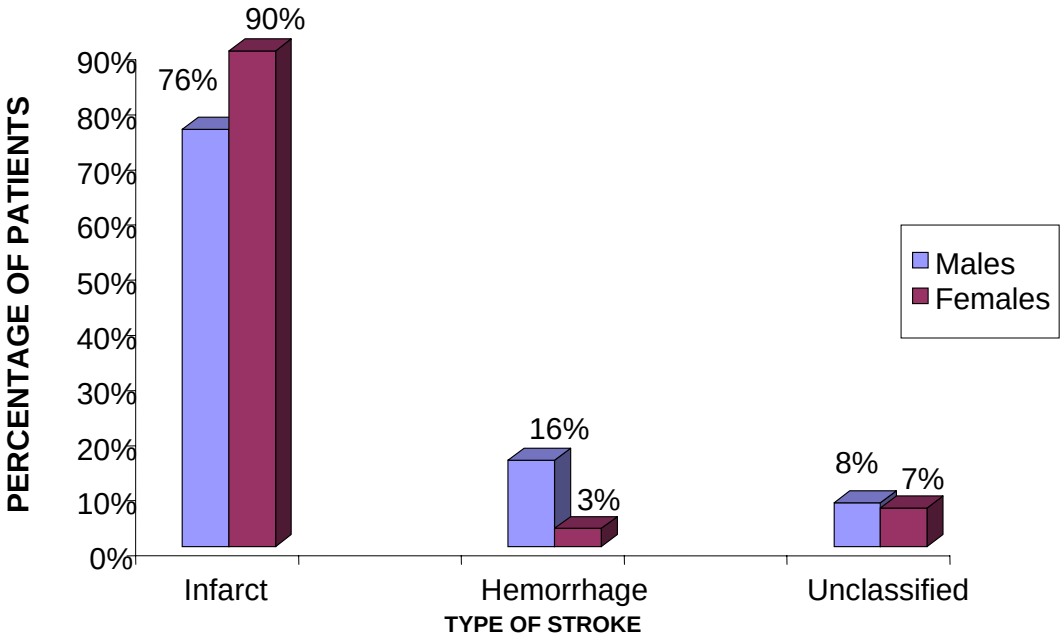
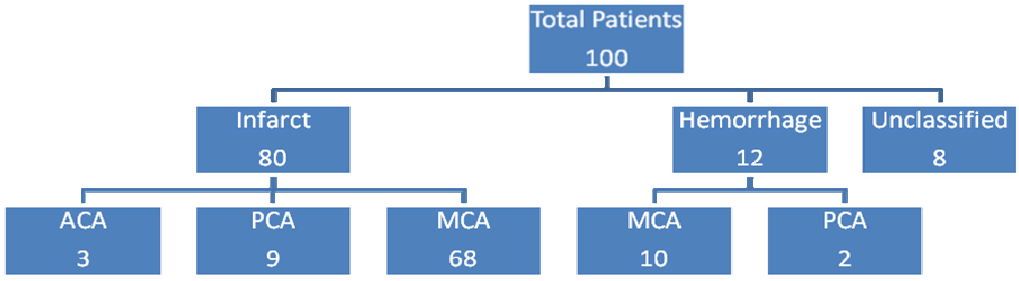


Chart 3: Territory Distribution in Stroke patients



ACA – Anterior Cerebral Artery,

MCA –Middle Cerebral Artery and

PCA – Posterior Cerebral Artery

Most common arterial territory involved in our study was **Middle Cerebral artery,** both in Thrombotic (85%) and Hemorrhagic (83.3%) patients followed by Posterior Cerebral artery (Thrombosis 11.3%; Hemorrhage 16.7%). Anterior Cerebral artery was not involved in hemorrhagic infarct.

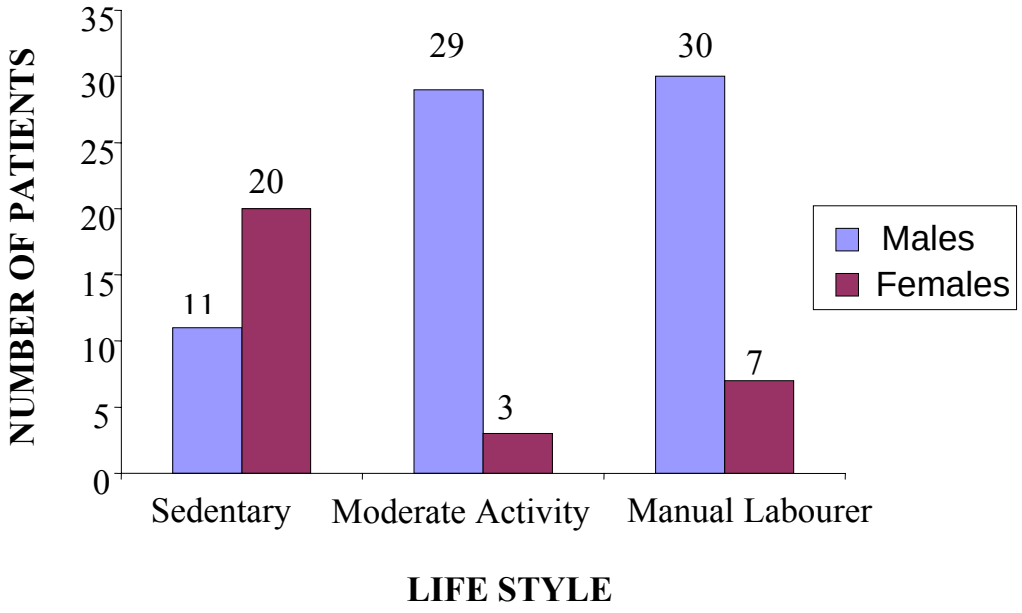
Table1: Gender in relation to stroke pattern and arterial territory involved

Territory	**Males**				**Females**				**Grand Total**
Infarct	Left	Right	Bilateral	Total	Left	Right	Bilateral	Total	
MCA	27	16	2	45	16	6	1	23	68
PCA	3	3	1	7	1	1	-	2	9
ACA	-	1	-	1	1	-	1	2	3
Unclassified				6				2	8
Subtotal	30	20	3	**59**	18	7	2	**29**	**88**
Hemorrhage									
MCA	3	7	-	10	-	-	-	-	10
PCA	1	-	-	1	1	-	-	1	2
ACA	-	-	-	-	-	-	-	-	-
Subtotal	4	7	-	**11**	1	-	-	1	**12**
Total Patients				**70**				**30**	**100**

- Most common arterial territory involved in both sexes with Ischemic stroke was **Left Middle Cerebral artery** (Male – 76.3%, Female 79.3%)

- In males with hemorrhage stroke, out of **11** patients, **10** had involvement of MCA territory and **1** had involvement of Posterior cerebral artery.
- Only one female had hemorrhage involving **Left Posterior territory.**

Chart 4: Occupation and Stroke



Out of 30 **female** stroke patients, **sedentary life style** contributed maximum **(66.7%)** to stroke whereas in 70 **males**, **manual labourers (42.9%)** had the maximum stroke incidence. Sedentary life style was associated with stroke risk only in 11 males (15.7%).

Fig – 5 CT BRAIN - PLAIN
LEFT FRONTO – PARIETO - TEMPORAL ISCHAEMIC INFARCT

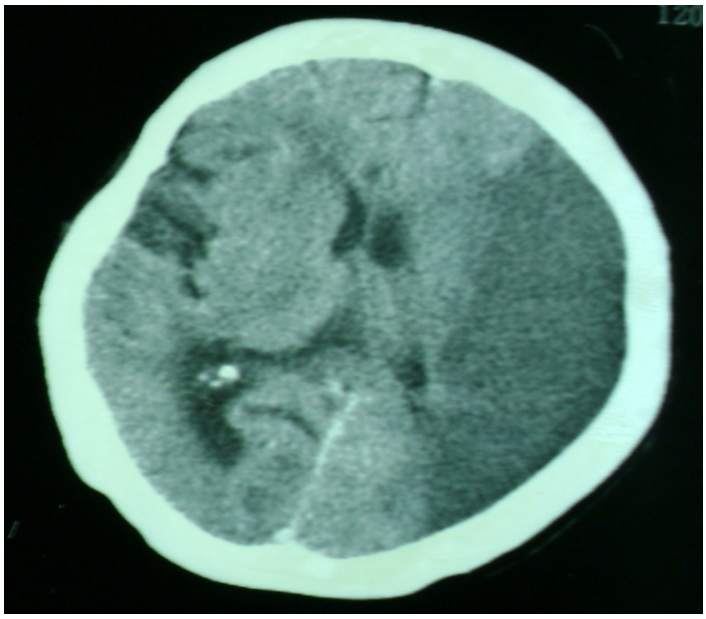


Fig – 6 CT BRAIN - PLAIN
LEFT PARIETO - TEMPORAL HEMORRHAGIC INFARCT WITH VENTRICULAR EXTENSION

Table 2: Occupation & Gender in relation to Stroke pattern

Stroke	Males				Females				Total
	Seden-tary	Moderate activity	Manual Labourer	Total	Sedentary	Moderate activity	Manual Labourer	Total	
Ischemic Stroke	7	23	23	53	19	2	6	27	**80**
Hemorrhage	2	4	5	11	0	1	-	1	**12**
Unclassified	2	2	2	6	1	-	1	2	**8**
Total	11	29	30	70	20	3	7	30	**100**

Cerebral hemorrhage was more common in Moderate activity workers and Manual labourers and least common in individuals leading sedentary life style. In 20 out of 30 females in our study, who lead sedentary life style, none had hemorrhage.

Table 3: Age wise and Gender wise stroke distribution

Age	Males	Females
≤ 40 years	9 (12.(%)	Nil
41-59 years	32 (45.7%)	8 (26.7%)
≥60 years	39 (55.7%)	22 (73.3%)

The most common age group affected by stroke in males was **≥ 60 years** (55.7%) followed by **41-59 years** (45.7%) whereas in females it was **≥ 60 years** (73.3%)

Chart 5- Age wise Male Stroke Patients

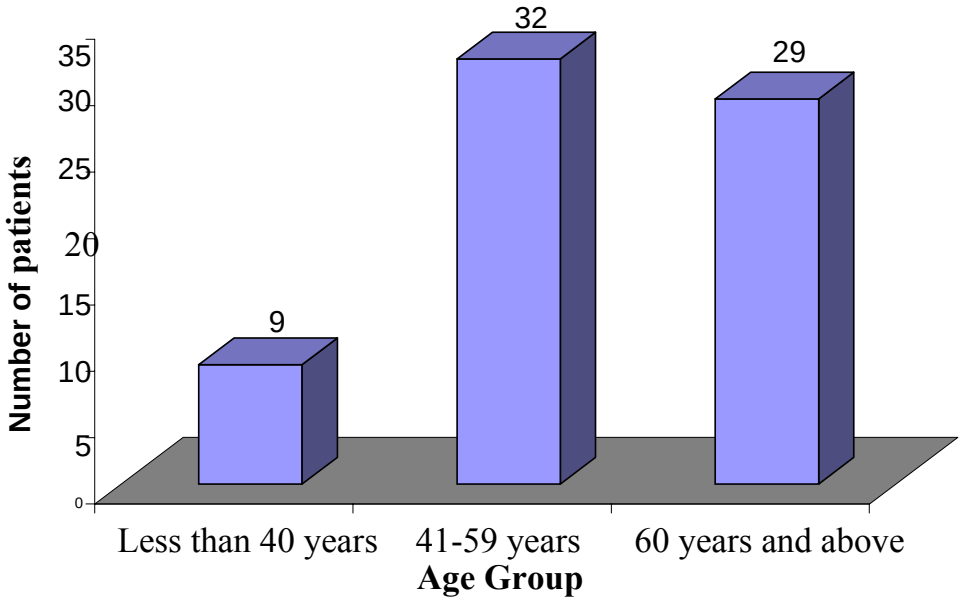


Chart 6 – Age wise Female Stroke Patients

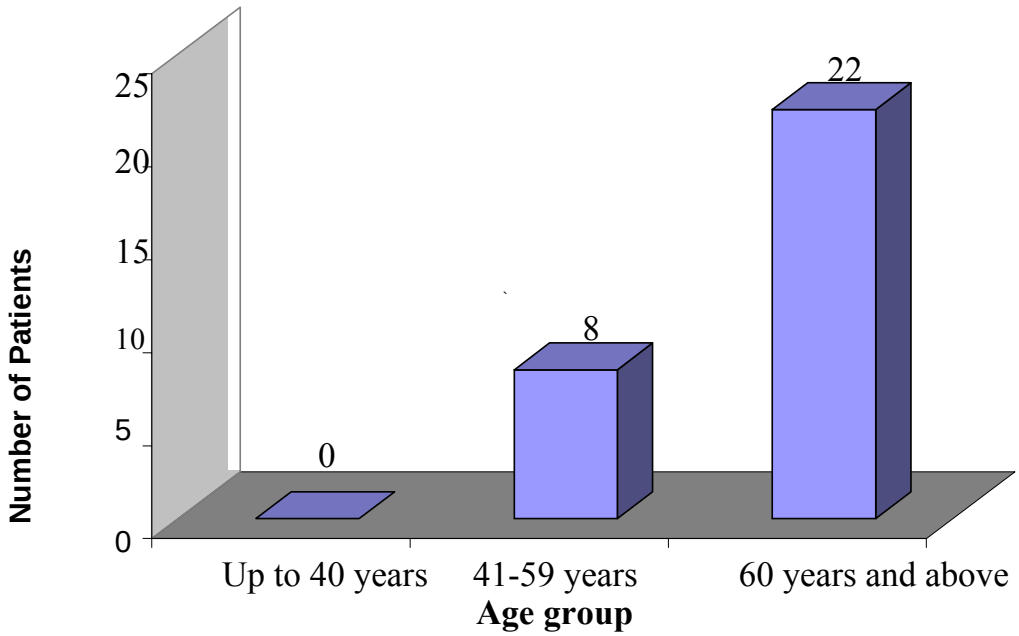


Table 4: Other Risk factors

	Males	**Female**	**Total**
1. Smoking	51	Nil	**51/70**
2. Alcohol	51	Nil	**51/70**
3. IHD	26/70 (37.1%)	9/30 (30%)	**35/100**
4. HTN (BP≥140/90mm Hg)	54/70 (77.1%)	24/30 (80%)	**78/100**
5. DM (FBG≥ 126 mg/dl)	12/70 (17.1%)	8/30 (26.7%)	**20/100**
6. Recurrent Stroke	5/70 (7.1%)	2/30 (6.7%)	**7/100**
7. Mean HDL (mg/dl)	38.6 ± 1.6	39 ± 2.9	-
8. Mean TGL (mg/dl)	140.6 ± 7	147 ± 9.8	-
9. Mean FBG (mg/dl)	111.5 ± 9.5	120 ± 18.8	-
10. Mean WC (inches)	31 ± 1.1	30 ± 1.4	-
11. Mean BP (mmHg)	153 ± 6.5/ 95 ± 4.5	156 ±10.2/ 90 ±5.8	-
12. Mean age (years) 13. TGL / HDL	56 ± 3 3.84 ± 0.3	63 ± 4.2 3.89 ± 0.4	-

Table 5: Age-wise distribution of various Metabolic Parameters in both Sexes

Table 5(i): Age-wise distribution of Fasting blood glucose (FBG) in both Sexes

	Males				Females			
Age	**No.**	**Mean FBG (mg /dl)**	**No. with FBG ≥ 100 mg/dl**	**%**	**No.**	**Mean FBG (mg /dl)**	**No. with FBG ≥ 100 mg/dl**	**%**
≤ 40 years	9	119.2	8	**88.9**	-	--	-	**Nil**
41-59 years	32	112	13	**40.6**	8	105	5	**62.5**
≥60 years	29	108.4	16	**55.2**	22	128	14	**63.6**

Irrespective of sex, all the age groups had **Mean Fasting blood glucose ≥100 mg/dl.** Out of **70** males and **30** females, **37** males and **19** females respectively had Fasting blood glucose ≥100 mg/dl**.** In males, the criterion of FBG for metabolic syndrome was most commonly fulfilled by age group **≤ 40 years (88.9%),** whereas in females, **> 40 years (63%)** group fulfilled the criteria.

Table 5 (ii): Age-wise distribution of HDL Cholesterol (HDL) in both sexes

	Males				Females			
Age	**No.**	**Mean HDL (mg /dl)**	**No. with HDL <40 mg/dl**	**%**	**No.**	**Mean HDL (mg /dl)**	**No. with HDL <50 mg/dl**	**%**
≤ 40 years	9	39.8	2	**22.2**	-	--	-	**Nil**
41-59 years	32	39.4	13	**40.6**	8	40	8	**100**
≥60 years	29	37.2	18	**62.1**	22	39.2	21	**95**

Out of **70** males, **33** had HDL < 40 mg/dl. Most common age group affected by HDL parameter was **≥60 years (62.1%).** In all the age groups, HDL was more or less the same, implying that it had not contributed in a major way for the metabolic syndrome.

Out of **30** females, **29** had HDL < 50 mg/dl. All females in the age group 41-**59 years** fulfilled the criteria for metabolic syndrome through impaired HDL levels (<50 mg/dl) followed closely by age group **≥60 years**. Irrespective of age group, females were at risk of metabolic syndrome due to impaired HDL levels (<50 mg/dl). It was also observed in all females that HDL was much lower than the desired level of 50 mg/dl.

Table 5 (iii): Age-wise Distribution of Triglyceride (TGL) in both sexes

	Males				Females			
Age	**No.**	**Mean TGL (mg /dl)**	**No. with TGL ≥ 150 mg/dl**	**%**	**No.**	**Mean TGL (mg /dl)**	**No. with TGL ≥ 150 mg/dl**	**%**
≤ 40 years	9	144.7	3	**33.3**	-	-	-	**-**
41-59 years	32	144.3	13	**40.6**	8	148	2	**25**
≥60 years	29	135.2	8	**27.6**	22	147	10	**45.5**

Out of **70** males, **24** had TGL ≥ 150 mg/dl. In all age groups, males had mean TGL level in the normal desired range (<150 mg/dl). Most common age group in which Triglyceride level fulfilled the criteria for metabolic syndrome was **41-59 years**. **(40.6%).**

Out of **30** females, **12** had TGL ≥ 150 mg/dl. All age-groups of females had mean TGL level in the normal desired range (<150 mg/dl). However, females in the age group ≥ **60 years (45.5%)** had high TGL levels (≥ 150 mg/dl) thereby fulfilling the criteria for metabolic syndrome.

Table 5 (iv): Age-wise Distribution of Waist Circumference (WC) in both sexes

	Males				Females			
Age	**No.**	**Mean WC (inches)**	**No. with WC ≥ 35 inches**	**%**	**No.**	**Mean WC (inches)**	**No. with WC ≥ 35 inches**	**%**
≤ 40 years	9	31.41	2	**22.2**	-	--	-	**Nil**
41-59 years	32	31	9	**28.1**	8	31	3	**37.5**
≥60 years	29	30.9	8	**27.6**	22	30	10	**45.5**

In all age groups, males had Mean Waist Circumference in the normal range of < 35 inches; whereas Females in the age group **41-59 years** had Mean Waist Circumference of **31 inches**. Males in the age group **> 41-59 years (28.1%) and ≥ 60 years (27.6%)** and Females in the age group **≥ 60 years (45.5%)** contributed most to the syndrome through the waist circumference parameter.

Table 5 (v): Age-wise distribution of Blood Pressure (BP) in both sexes

	Males				Females			
Age	**No.**	**Mean BP (mmHg)**	**No. with BP ≥ 130/85 mmHg**	**%**	**No.**	**Mean BP (mmHg)**	**No. with BP ≥ 130/85 mmHg**	**%**
≤ 40 years	9	156.2/ 90.8	6	**66.7**	-	--	-	**Nil**
41-59 years	32	150.2/ 93.1	23	**71.9**	8	158 / 99	7	**87.5**
≥60 years	29	169.4/ 98.3	26	**89.7**	22	158 / 96	18	**81.8**

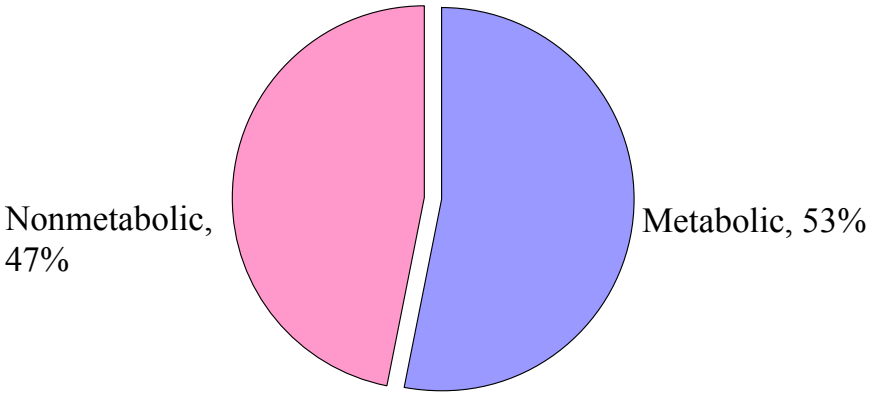
Irrespective of age and sex, all had mean BP in the hypertensive range according to Stage I of JNC VII (140-159 / 90-99 mm Hg). Out of **70** males, **55** had BP ≥ 130/85 mm Hg and the most affected were in the age group **≥ 60 years (89.7%)**

Out of **30** females **≥ 40 years, 25** had metabolic syndrome due to BP criteria of ≥130 / 85 mmHg.

Table 6: Prevalence of Metabolic Syndrome in Stroke Patients

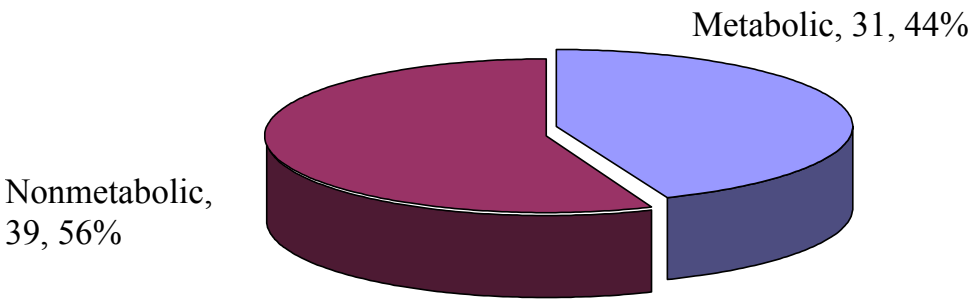
Sex	**Number of Patients**	**Number of Metabolic Patients**	**%**
Male	70	31	**44.3**
Female	30	22	**73.3**

Chart 7 - Prevalence of Metabolic syndrome in Stroke Patients



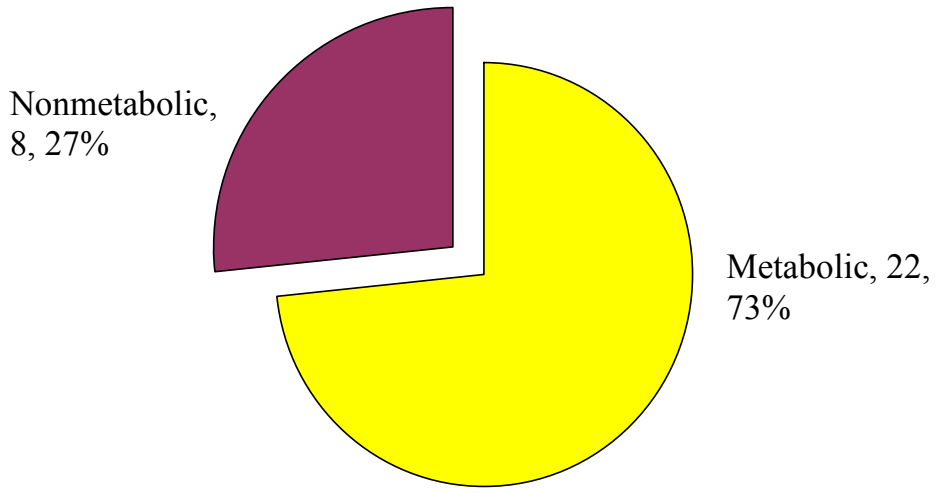
Out of **100** stroke patients under the present study, **53** (**31** males and 22 females) fulfilled the criteria for metabolic syndrome.

Chart 9 - Prevalence of Metabolic syndrome in Males (n=70)



Out of the **70** male patients, **31** patients had metabolic syndrome and **39** were non –metabolic.

Chart 9 - Prevalence of Metabolic syndrome in Females (n=30)



Out of the **30** females, **22** had metabolic syndrome and 8 were non-metabolic.

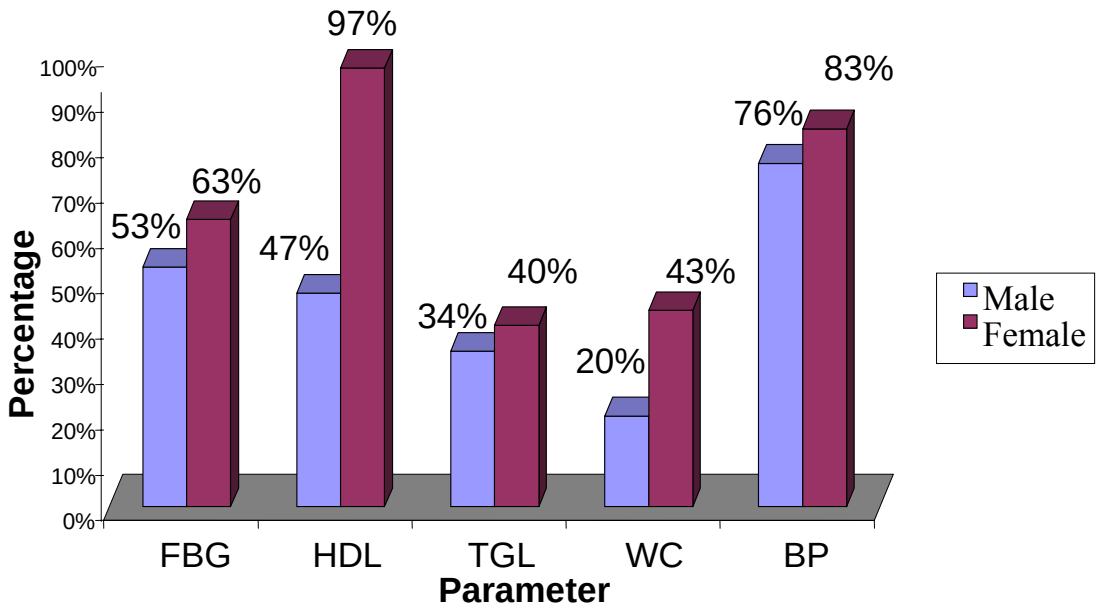
Table 7: Metabolic Syndrome and Type of stroke

Type of Stroke	Male Metabolic n=31	Female Metabolic n=22	Total n=53
Ischemic	23	20	**43**
Hemorrhage	7	1	**8**
Unclassified	1	1	**2**

In both sexes with metabolic syndrome, **Ischemic stroke** was the most Common stroke type observed (43 patients i.e. **81%**).

Chart10: Percentage Contribution of each metabolic parameter to Metabolic syndrome in both Sexes.

No. of males-**70**, females-**30**



FBG - Fasting Blood Glucose

HDL- High Density Lipo protein

TGL – Triglyceride

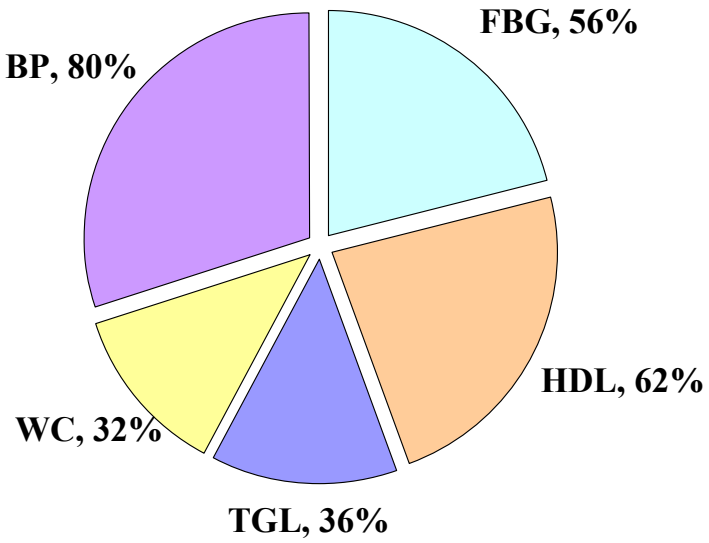
BP- Blood Pressure,

WC - Waist Circumference

- **19 (63%)** females and **37(53%)** males had FBG ≥ 100 mg/dl that fulfilled the syndrome defining criteria. (Overall contribution of **56%** by FBG to the syndrome.)
- **29 (97%)** females and **33** (**47%)** males contributed to the syndrome due to abnormal HDL levels.

- **12 (40%)** females and **24 (34%)** males had abnormal TGL levels (≥ 150 mg/dl).
- **13 (43%)** females and **19 (20%)** males fulfilled the criteria by having abnormal waist circumference.
- Abnormal BP (≥ 130/85) as the metabolic parameter was observed in **25 (82%)** females and **55 (76%)** males.

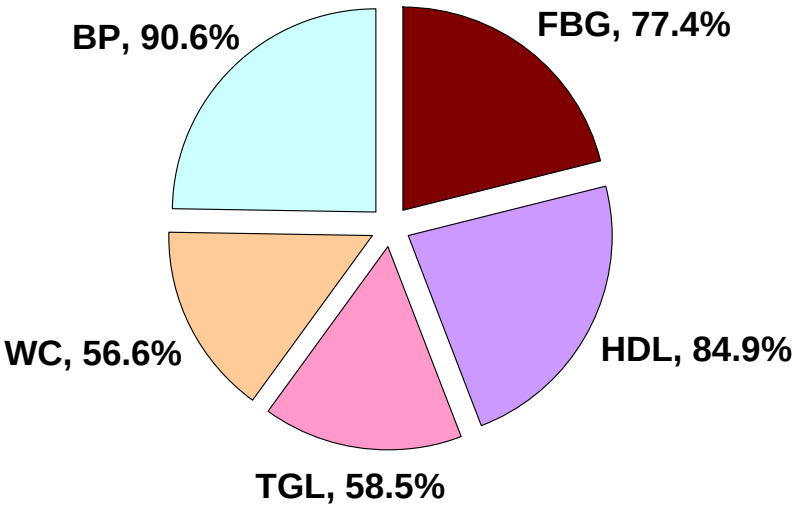
Chart 11: Percentage Contribution of each metabolic parameter in 100 stroke patients



FBG - Fasting Blood Glucose
HDL-High Density Lipo protein
TGL – Triglyceride
BP- Blood Pressure,
WC - Waist Circumference

Most common parameter that contributed the maximum to Metabolic syndrome was **Blood Pressure (80%).**

Chart 12: Percentage Contribution of each metabolic parameter in 53 metabolic syndrome patients



FBG - Fasting Blood Glucose

HDL-High Density Lipo protein

TGL – Triglyceride,

WC - Waist Circumference

BP- Blood Pressure

Most common parameter that contributed the maximum to Metabolic syndrome was **Blood Pressure (90.6%).**

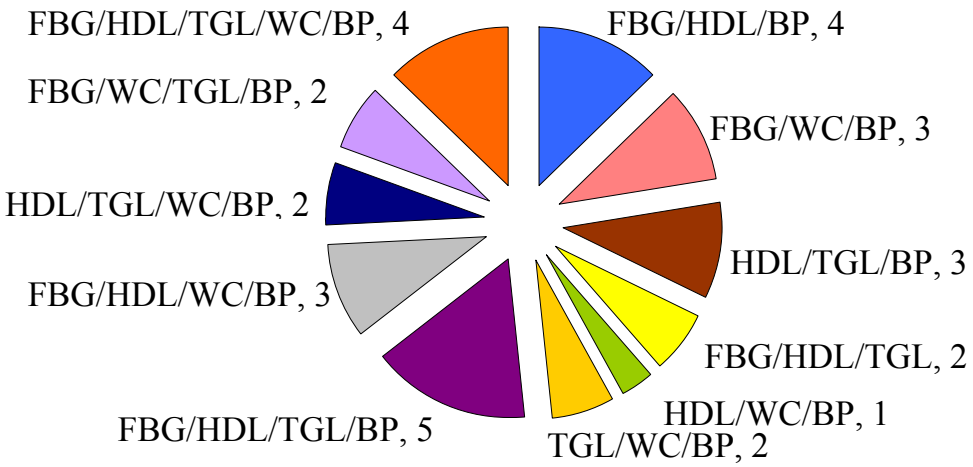
Table 8: Metabolic syndrome and age in both sexes

Age (years)	Female Patients	No. of Metabolic female patients	%	Male Patients	No. of Metabolic male patients	%
≤ 40	--	--	--	9	3	33.3
41-59	8	5	62.5%	32	14	43.8
≥ 60	22	17	77.3%	29	14	48.3

In females, most common age group affected by Metabolic syndrome was **≥ 60 years** (77.3%) whereas in males 43.8 % of **41-59 years group** and 48.3% of ≥ had larger number of metabolic syndrome patients.

Overall, metabolic syndrome was more prevalent in females **(> 40 years**) than in males.

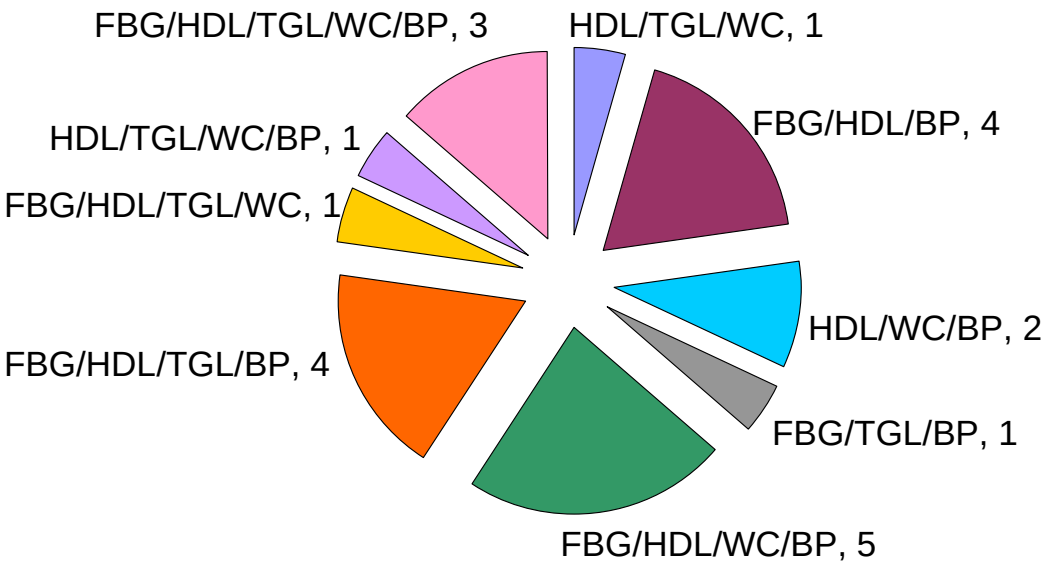
Chart 13: Distribution of Various Parameters in Male Metabolic syndrome Patients (N=31)



FBG - Fasting Blood Glucose
HDL-High Density Lipo protein
TGL – Triglyceride,
WC - Waist Circumference
BP- Blood Pressure

Out of the total **31** Metabolic Syndrome male patients, **15 (48.4%)** patients were with 3 parameters, **12 (38.7%)** were with 4 parameters and **4 (12.9%)** were with all 5 parameters.

Chart 14: Distribution of Various Parameters in Female Metabolic syndrome Patients (N=22)



FBG - Fasting Blood Glucose
HDL-High Density Lipo protein
TGL – Triglyceride,
WC - Waist Circumference
BP- Blood Pressure

Out of the total **22** Metabolic Syndrome female patients, **8 (36.4%)** patients were with 3 parameters **-11 (50%)** were with 4 parameters and **3 (13.6%)** were with all 5 parameters.

Table 9: Smoking and Metabolic syndrome

	Smoking	**Non-smoking**	**Total**
Metabolic syndrome	19	12	31
Non Metabolic	32	7	39
Total	**51**	**19**	70

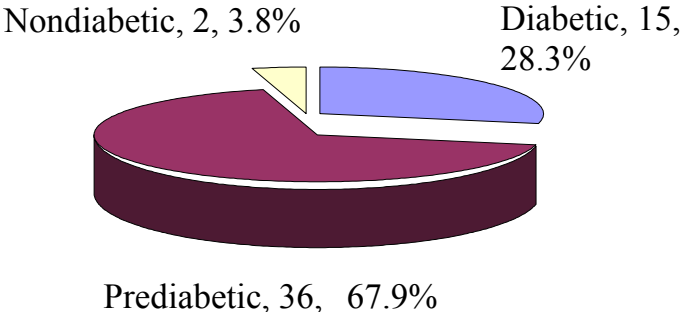
None of the females in our study had the habit of smoking. Out of the **51** smokers in males, **19 (37%)** had metabolic syndrome. Out of the **19** nonsmokers, **12 (63.2%)** had metabolic syndrome.

Table 10: Alcohol and Metabolic syndrome in males

	Alcoholic	**Non-alcoholic**	**Total**
Metabolic syndrome	23	8	31
Non Metabolic	29	10	39
Total	**52**	**18**	70

None of the females under our study were alcoholic. Of the **70** males, **52** were alcoholic and within them **23(44%)** had metabolic syndrome. Of the **18** nonalcoholic males, **8 (44%)** had metabolic syndrome.

Chart 15: Diabetes Mellitus and Metabolic syndrome (N=53)



Out of 53 metabolic syndrome patients, **15(28.3%)** were diabetics & **36 (67.9%)** were prediabetics.

Table 11: Distribution of Diabetes and Prediabetes in Metabolic syndrome population

Diabetic- **FBG ≥126 mg /dl**

Prediabetic (impaired fasting glucose) - **FBG 100 -125 mg /dl**

	Males		**Females**	
	Diabetic	**Prediabetic**	**Diabetic**	**Prediabetic**
Metabolic	7 / 31	16 / 31	8 / 22	10 / 22
Nonmetabolic	5 / 39	9 / 39	Nil	1 / 8
Total	**12 / 70**	**25 / 70**	**8 / 30**	**11 / 30**

Out of **70** male stroke patients, **12 (17.1%)** were diabetic and **25 (35.7%)** were prediabetic. Out of **31** male metabolics, **7 (22.6%)** were diabetic and **16 (51.6%)** were prediabetic.

Out of the **12** diabetic males, **7 (58.3%)** had metabolic syndrome and out of **25** prediabetic males, **16 (51.6%)** had metabolic syndrome.

Of the **30** female stroke patients, **8 (26.7%)** were diabetic and **11 (36.7%)** were prediabetic. Out of 22 female metabolic, **8 (36.4%)** were diabetic and **10 (45.5%)** were prediabetic. While all 8 diabetic women had metabolic syndrome, only **10 (91%)** out of 11prediabetic women, had metabolic syndrome.

Table 12: Hypertension and Metabolic syndrome

Hypertension - (≥ 140 /90 mm Hg- Stage I according to JNC VII)

Prehypertension- (Systolic 121-139 mmHg and Diastolic 81-89 mm Hg)

	Males		**Females**	
	Hypertensive	**Prehypertensive**	**Hyper-tensive**	**Prehyper-tensive**
Metabolic	29 / 31	--	19 / 22	1 / 22
Nonmetabolic	25 / 39	7 / 39	5 / 8	1 / 8
Total	**54 / 70**	**7 / 70**	**24 / 30**	**2 / 30**

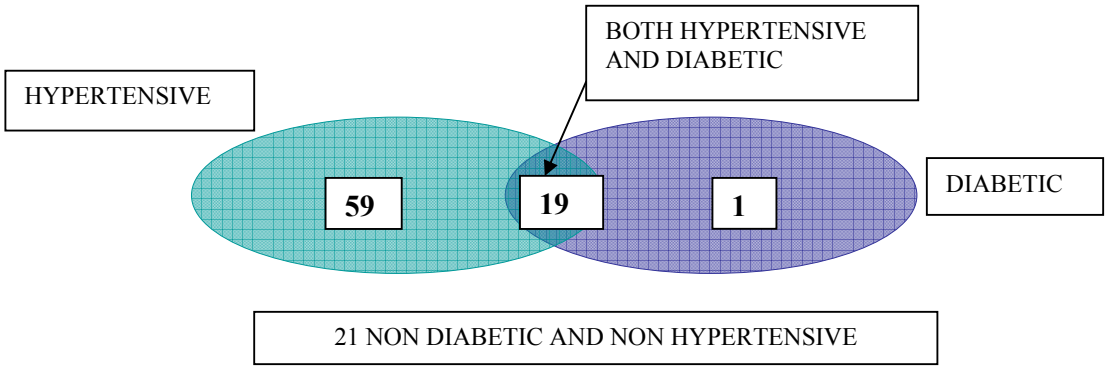
Out of **70** male stroke patients, **54 (77.1%)** were Hypertensive and **7 (10%)** were Prehypertensive. Out of **30** female stroke patients, **24 (80%)** were Hypertensive and **2 (6.7%)** were Prehypertensive. Out of the total **53** metabolic syndrome patients, **48** (29 males and 19 females) **(90.6%)** were hypertensive.

Out of **31** male metabolics, **29 (93.5%)** were Hypertensive and none were Prehypertensive. Out of **22** female metabolics, **19 (86.4%)** were Hypertensive and **1 (4.5%)** was Prehypertensive.

Out of **54** hypertensive males, **29 (53.7%)** had metabolic syndrome. Out of **24** hypertensive females, **19 (79.2%)** had metabolic syndrome and out of **2** Prehypertensive females, **1 (50%)** had metabolic syndrome**.**

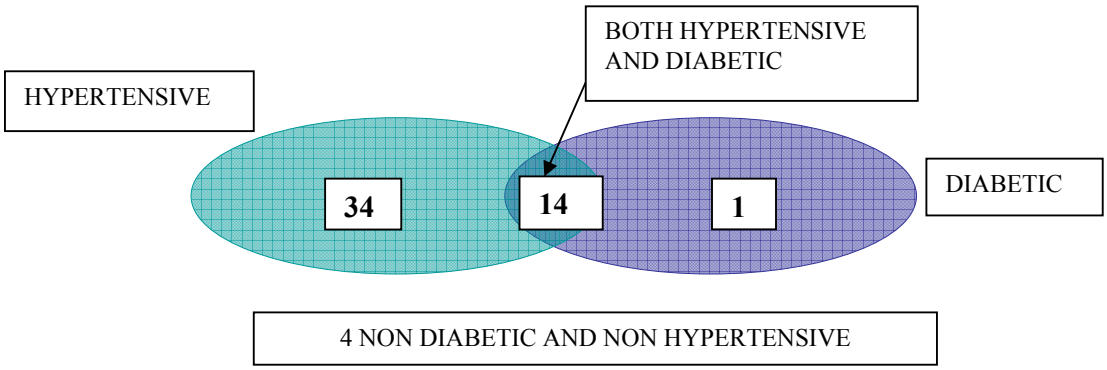
Chart 16: Both Hypertension and Diabetes in combination with Metabolic syndrome

a) N=100 stroke patients



Out of 100 stroke patients under our study, **19** had both Diabetes and Hypertension.

b) N=53 metabolic syndrome patients



Out of 53 metabolic syndrome patients, **14 (26.4%)** had both hypertension and diabetes.

Table 13: HDL Cholesterol and Metabolic Syndrome

	Males		Females	
	No. with HDL<40 mg / dl	**No. with HDL≥40 mg / dl**	**No. with HDL<50 mg / dl**	**No. with HDL≥ 50 mg / dl**
Metabolic	24 / 31	7 / 31	21 /22	1 / 22
Nonmetabolic	9 / 39	30 / 39	8 / 8	Nil
Total	**33 / 70**	**37 / 70**	**29 / 30**	**1 / 30**

Out of **31** male metabolics, **24 (77.4%)** had **HDL<40 mg / dl.** Out of **30** female stroke patients, **29(96.7%)** had **HDL< 50 mg / dl** and remaining **1(3.3%)** had HDL in the normal range. Out of these **29** females with **HDL< 50 mg / dl**, **21(72.4%)** had metabolic syndrome. Out of **22** female metabolics, **21 (95.5%)** had **HDL< 50 mg / dl** and all the **8** non-metabolic patients had **HDL< 50 mg / dl.**

Table 14: Triglyceride (TGL) and Metabolic Syndrome

	Males		**Females**	
	No. with TGL≥150 mg / dl	**No. with TGL<150 mg / dl**	**No. with TGL≥150 mg / dl**	**No. with TGL<150 mg / dl**
Metabolic	20 / 31	11 / 31	11 /22	11 / 22
Nonmetabolic	4 / 39	35 / 39	1/ 8	7 / 8
Total	**24 / 70**	**46 / 70**	**12 / 30**	**18 / 30**

Out of **70** male stroke patients**, 24 (34.31%)** had **TGL≥150 mg / dl** and remaining **46 (65.7%)** had TGL in the normal range. Out of **24** males with **TGL≥150 mg / dl**, **20 (83.3%)** had metabolic syndrome and out of **46** males with normal TGL (<150 mg / dl), only **11 (23.9%)** had metabolic syndrome. Out of **31** male metabolics, **20 (64.5%)** had TGL**≥150 mg / dl.**

Out of **30** female stroke patients, **12 (40%)** had **TGL≥150 mg / dl** and remaining **18 (60%)** had TGL in the normal range. Out of these **12** females with **TGL≥150 mg / dl**, **11(91.7%)** and out of **18** females with normal TGL (<150 mg / dl), **11 (61%)** had metabolic syndrome. Out of **22** female metabolics, **11 (50%)** had **TGL≥150 mg / dl.**

Table 15: Waist circumference (WC) and Metabolic Syndrome

	Males		**Females**	
	No. with WC ≥35 inches	**No. with WC < 35 inches**	**No. with WC ≥ 31inches**	**No. with WC < 31inches**
Metabolic	17 / 31	14 / 31	13 /22	9 / 22
Nonmetabolic	2 / 39	37 / 39	Nil	8 / 8
Total	**19 / 70**	**51 / 70**	**13 / 30**	**17 / 30**

Out of **70** male stroke patients**, 19(27%)** had **WC≥35 inches** and remaining **51(72.9%)** had WC<35 inches. Out of 19 males with WC≥35 inches, **17 (89.5%)** had metabolic syndrome and out of **51** males with WC<35 inches, only **14 (27.5%)** had metabolic syndrome. Out of **31** male metabolics, **17 (54.8%)** had **WC ≥35 inches.**

Out of **30** female stroke patients, **13 (43.3%)** had **WC≥ 31inches** and remaining **17 (56.7%)** had WC< 31inches. Out of these **13** females with **WC≥ 31inches,** all **13 (100%)** and out of **17** females with WC< 31inches, **9 (52.9%)** had metabolic syndrome. Out of **22** female metabolics, **13 (59.1%)** had **WC≥ 31 inches.** All non metabolic females had normal waist circumference.

Discussion

DISCUSSION

Lipid profile & Stroke

In a case control study conducted on 76 patients, with an age range of 40-70 years by ***Immanuel Sin***[35]**,** consisting of 38 post ischemic stroke patients and 38 controls, HDL levels were significantly lower in stroke patients as compared to control subjects ($p<0.05$). The study concluded that low HDL-cholesterol level is a risk factor for ischemic stroke, with an odds ratio of 3.09, while total cholesterol, triglyceride and high LDL-cholesterol levels were not risk factors for ischemic stroke.

In another cohort study of 10 years follow up, by Kurth T ***et al***[32] in 27,937 US women aged $\geq$ 45 years, to evaluate the association between various lipid parameters and the risk of ischemic stroke, it was seen that low HDL, LDL, Total cholesterol& TC/HDL ratio were significantly associated with increased risk of ischemic stroke.

In the present study, low HDL (47% in males & 96.7% in females) was more strongly associated with ischemic stroke than high TGL (34.3% in males & 40% in females).

Prevalence of metabolic syndrome

Shrestha et al[37]	-	32%
E.I. Sorkhou et al[40]	-	34%
Milionis HJ et al &others[33]	-	46%
Present study	-	**53%**

In a cohort study done by Kurl S ***& et al***[30] in 1131men, patients with the metabolic syndrome as defined by the NCEP criteria had a 2.05-fold risk for all strokes and 2.41-fold risk for ischemic stroke, after adjusting for socioeconomic status, smoking, alcohol and family history of coronary heart disease.

In a cross-sectional analysis done by ***V. Athyros et al and others***[36] in 9669 Greek adults, the age-adjusted CVD prevalence was compared in metabolic syndrome patients according to NCEP-ATP-III, IDF and AHA/NHLBI criteria. It was shown that CVD prevalence increased in the presence of metabolic syndrome irrespective of the definition used. However, this increase was more pronounced when the NCEP-ATP-III and AHA/NHLBI criteria were implemented compared with the IDF definition.

Sex and metabolic syndrome

In a study done by ***Hillier TA et al***[39] in older women (≥ 65 years) with diabetes, metabolic syndrome was associated with two- to threefold higher mortality risk due to CVD. This is in accordance with this study where 64% females above 60 years fulfilled the criteria for stroke. Further, several studies have shown females to have higher prevalence rate of metabolic syndrome than males in accordance with this study (73% vs. 44%).

Metabolic parameters and Stroke

In a prospective cohort study done by Koren-Morag N ***et al***[29] in 14,284 patients with atherosclerotic cardiovascular disease to find relation between the metabolic syndrome and ischemic stroke or transient ischemic attack ,3703 (26%) had metabolic syndrome without diabetes and 3500(25%) had frank diabetes alone. The study showed that all components of the metabolic syndrome were associated with increased risk for ischemic stroke or TIA, but impaired fasting glucose and hypertension were the strongest risk predictors.

In a study by ***Wolf et al***[6] in 2097subjects, between 51-80, 22% met the criteria for metabolic syndrome alone, 5% for diabetes alone and 5% had both conditions.

In comparison, in this study, **23%** met the criteria for metabolic syndrome alone, **10.6%** for diabetes alone, and **28%** had both conditions.

In a cohort study done by ***Najarian et al***[34] in 2097 patients including 216 diabetic in Boston, it was found that 27.6 percent of the men and 21.5 percent of the women met the criteria for a diagnosis of metabolic syndrome without including IFG (IFG-126 mg %) patients. When 216 IFG individuals were included in the analysis, 30.3 percent of men and 24.7 percent of women met diagnosis criteria. The study concluded that metabolic syndrome and type 2 diabetes are independent risk factors for stroke. It was predicted that getting rid of their metabolic syndrome would eliminate about 20 percent of all strokes.

In comparison, the present study results show that **26%** of the men and **18%** of the women met the criteria for a diagnosis of metabolic syndrome without including glucose criteria (FBG >= 100 mg%) which increased to 74.2% & 81.8% in men & women respectively after including the glucose criteria. This huge increase in the prevalence of metabolic syndrome after applying the glucose criteria in our study is probably because of new modified definition of the syndrome used in the

study & also depicting the more potent contribution of prediabetes (IFG &IGT) to metabolic syndrome than other criterion factors.

In a study by ***Gupta and co-workers***[31] which included 19,257 hypertensive patients, 8091 (42%) were identified as having the metabolic syndrome at baseline according to the modified Adult Treatment Panel III (ATP III) criteria. When the individual components of the metabolic syndrome were adjusted, the metabolic syndrome was associated with a significantly increased risk for stroke as well as all-cause mortality (p=0.03 and p=0.04, respectively).

In contrast, in this study, out of 78 hypertensives, 48 **(61.5%)** had metabolic syndrome.

In a study done by W**.** ***Lee et al and others***[38] using the Asia–Pacific criteria for abdominal obesity based on waist circumference (APC–WC: ≥90cm in men, ≥80cm in women), prevalence rates of metabolic syndrome were 10.9% (9.8% male, 12.4% female), The age-specific prevalence of the metabolic syndrome increased in both male and female participants, and females had higher prevalence rates than males in age groups older than 50 years.

In contrast, in our study, prevalence rates based on waist circumference were **30 %(27% male, 43% female).**

Summary

SUMMARY

1) Ischemic stroke is the most common stroke pattern seen in Indian adults, more prevalent in males than in females; **80%** being thrombotic episodes as compared to **12%** hemorrhagic ones.
2) The most common territory involved in both ischemic and hemorrhagic stroke was middle cerebral artery **(85% and 16.7%** respectively).Anterior cerebral artery was not involved in hemorrhagic stroke in this study.
3) In both sexes, ischemic stroke had predilection for left middle cerebral artery **(63%)** whereas right middle cerebral artery (**32%)** was most frequently involved in hemorrhagic stroke.
4) In females, sedentary life style **(67%)** was the most common contributory factor to stroke whereas in males, manual labourers were affected the most by stroke.
5) In both sexes, stroke was most common in the age group≥60 years (male-**56%**; females-**73%**).
6) Mean age of stroke presentation in this study was **56± 3 years** in males and **63 ± 4.2 years** in females signifying earlier onset of risk factors in males as compared to females.
7) The prevalence of hypertension in stroke patients was high (**77%** males, **80%** females).

8) The prevalence of diabetes (FBG ≥126 mg/dl) in stroke patients was higher in females **(27%)** than in males **(17%).**

9) Mean triglyceride (**140.6 ± 7** mg/dl in males,**147 ± 9.8** mg/dl in females) and waist circumference (**31± 1.1 inches** in males,**30± 1.4 inches** in females) were in the normal range in both sexes, whereas mean HDL cholesterol was well below the normal desired range (**38.6± 1.6 mg/dl** in males,**39± 2.9mg/dl** in females).

10) In both sexes, mean blood pressure was in the stage I hypertensive range and mean fasting blood glucose was in the prediabetic range of 100-125 mg/dl suggesting that both hypertension and diabetes are independent strong risk factors for stroke and prediabetics are prone for macro vascular complications even before they are labeled as diabetics.

11) Most common age group affected in males with FBG≥ 100 mg/dl was **≤ 40 years** (89%).

12) Most frequently affected age group by low HDL cholesterol and high blood pressure in males was **≥ 60 years** (62% & 90 %respectively) and the same in females was **41-59 years** (100% and 88% respectively) emphasizing the high prevalence rate of metabolic syndrome in this age group.**41-59 years** was the most affected age group due to triglyceride (41%) and waist circumference (28%) parameters.

13) In females, the age group most frequently affected by FBG ≥ 100 mg/dl, Waist circumference and Triglyceride was **≥ 60 years** (64%, 46%& 46 % respectively).

14) Overall the prevalence of Metabolic syndrome was **53%**.It was more prevalent in females **(73%)** than in males **(44%)** & all the 5 parameters affected them more than males.

15) Overall, blood pressure was the parameter that contributed most **(80%)** to metabolic syndrome followed by HDL cholesterol **(62%)** and FBG **(56%)** in that order.

16) The parameter that contributed most to metabolic syndrome was high blood pressure in males and low HDL cholesterol in females.

17) In this study, none of the females smoked or consumed alcohol. In males, **73%** were smokers and **74%** were alcoholics; but only 37% and 44% respectively had metabolic syndrome. Further, 63% non smokers and 44% non alcoholics had metabolic syndrome. This implies that though smoking and alcohol are important risk factors for stroke, they don't contribute to metabolic syndrome in a significant way.

18) Ischemic stroke was the most common stroke pattern observed in metabolic syndrome patients.

19) Prevalence of both diabetes & hypertension was seen in 19% stroke patients which increased to 26% in metabolic syndrome patients signifying the consistent strong association between these two factors & their strong contribution to the syndrome.

20) HDL Cholesterol significantly contributed to the syndrome in both sexes (**73%** in males & **72%** in females). Also, % of metabolic syndrome patients associated with abnormal HDL levels was also significant (**77%** in male metabolics &**96%** in female metabolics). All females with normal HDL levels were non metabolic and only 19% males with normal HDL levels had metabolic syndrome. So both HDL and metabolic syndrome were strongly associated with each other.

21) Triglyceride levels significantly contributed to the syndrome in both sexes (**83%** in males & **92%** in females).Also, metabolic syndrome was associated significantly with high triglyceride in both sexes (65% in males &50 % in females). But 24% males &61% females with normal TGL levels conversely had metabolic syndrome. Both high TGL & Metabolic syndrome were associated with each other but not as strong as low HDL & high blood pressure.

22) Waist circumference significantly contributed to the syndrome in both sexes (**90%** in males & **100%** in females). Also, % of metabolic syndrome patients associated with abnormal waist circumference was significant in both sexes (55% in males &60 % in females). But 28% males & 53% females with normal waist circumference had metabolic syndrome. All female patients with abnormal waist circumference had metabolic syndrome and all non-metabolics had normal waist circumference showing strong association between waist circumference and metabolic syndrome in them.

Conclusion

CONCLUSION

- Ischemic stroke with involvement of left middle cerebral artery is the most common stroke seen in Indian adults, affecting most commonly sedentary females and male manual labourers in the age group $\geq$ 60 years.
- Hypertension, Diabetes and low HDL levels are the most important risk factors for stroke, more so in females.
- Males in age group 41-59 years are at increased risk of metabolic syndrome due to all parameters a little earlier than females who are at risk above 60 years.
- Indians have high prevalence rate of metabolic syndrome (53%) affecting females more than males.
- Blood pressure is the most common contributing factor to the syndrome.
- All factors significantly contribute to the syndrome but metabolic syndrome is strongly associated with high blood pressure, impaired fasting glucose and low HDL levels only, except in females in whom the syndrome & waist circumference are strongly associated.

Annexure

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Proforma

PROFORMA

STUDY OF METABOLIC SYNDROME IN STROKE PATIENTS (NCEP ATP III MODIFIED CRITERIA)

NAME :

AGE :

SEX : **MALE / FEMALE**

ADDRESS:

OCCUPATION : **SEDENTARY**
MODERATE ACTIVITY
MANUAL LABOURER

RISK FACTORS :

1. FASTING BLOOD GLUCOSE (mg/dl)

2. FASTING LIPID PROFILE **HDL-C (mg/dl):**
TGL (mg/dl):

3. WAIST CIRCUMFERENCE (inches):

4. HIP MEASUREMENT:

5. WAIST HIP RATIO:

6. BODY MASS INDEX: **WEIGHT:** **HEIGHT:**

7. BLOOD PRESSURE (mmHg):

8. ECG:

9. ECHO:

10. SMOKING; **YES / NO**

11. ALCOHOL: **YES/NO**

12. ACANTHOSIS NIGRICANS YES/NO

13: CT BRAIN

DIAGNOSIS: **ISCHEMIC**
HEMORRHAGIC
UNCLASSIFIED

TREATMENT:

OUTCOME **DEATH / RECOVERY**

NOTE:
OCCUPATION

Sedentary	-	Unemployed at home, Office workers, Professional.
Moderate activity	-	Semiskilled and skilled professionals like weavers, drivers, workshop - worker, cobbler, salesman, broker, carpenter etc
Manual labourers	-	Unskilled daily wagers, farmers, building construction workers etc.